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Original Articles

THE EVOLUTION AND DEVOLUTION OF THE HUMAN FACE

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IN THE evolution of higher mammals, as well as in that of lower vertebrates, the face is preponderant in size over the exiguous brain case. For the face is the seat of the principal sense organs that serve as the animal's media of communication with the outside world, in addition to the orifices of the nutritive and respiratory intakes. The central nervous system is, of course, lodged principally in the skull, but the size of the brain essential for sensory and motor functions in all but the highest primates is small relative to facial dimensions and body bulk, and seems to diminish as the animals get larger. An indispensable minimum of brain size for the nervous functioning of smaller mammals seems to result in a relatively much larger brain case as compared with the face than is usually found in closely related animals that are bigger, later, and, in many respects, higher evolutionary products. Thus the size of the brain in the marmoset, the most primitive and one of the smallest monkeys, is big enough to give it the most humanoid profile in all of the primates below man. On the other hand, the giant gorilla, which is vastly superior to the marmoset in evolutionary status, has a face and jaws that dwarf his brain case.

Gregory, in one of his masterly works on the evolution of the face and the dentition,† points out that the face owes its indispensability to its primary concern with (1) "detection of desirable sources of energy, (2) direction of locomotor machinery toward its goal, (3) capture and preliminary preparation of energy-giving food," and, among higher animals, its function (4) "as a lure for the capture of a mate." (However, in this lecture I am not going to ex-

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†Gregory, William K.: Our Face From Fish to Man, 1929, pp. 3, 4.

patiate upon the problem of the orthodontist in creating facial allure in repellent physiognomies, however rewarding the solution of that task may be both to patient and to practitioner.)

Although our interest is concentrated upon the later stages of evolution and devolution of the human face, we must recapitulate the early phases of its development for historical purposes and in order to invest our discussion with that haze of scientific profundity which applied scientists expect of an anthropologist or any other impractical theorist.

The notorious shark, a byword in lower vertebrate evolution (as well as in lower finance) has, as Gregory remarks, a wedge-shaped brain case which serves as a thrust block to receive the forward push from the backbone and the reaction from the water. Thus it may be seen that the primitive brain case in the aquatic vertebrate is a species of prow, that "prowess" means getting ahead, and that the individual who butts his head against something or even "butts in" is behaving according to the best traditions of his lower vertebrate ancestry.

Initially, it would appear that where the head is going and what it is going up against are more important than what is going on inside the head. You are familiar, of course, with the fact that the shark does not even possess a fully ossified skeleton; that its jaws are cartilaginous, its jaw muscles merely modified gill-arch muscles, and its teeth specializations of the tough shagreen or skin that is studded with minute calcified papillae or denticles. You know too that successive sets of these flat, triangular, saw teeth come into use as they are worn down.*

In the ancient lobe-finned, air-breathing, ganoid fishes, which are much nearer the line of human evolution than the shark, the skin of the face and over the skull is stiffened into a bony mask by the deposition of phosphate of lime, and the whole surface of the mask is covered by a thin, enamel-like layer. This mail-clad face and skull are retained in the oldest known fossil amphibians, the earliest four-footed, air-breathing, land-living forms. It is a starting point for the 28 bones retained in the human skull after ages of reduction and elimination of superfluous bony elements. In the amphibians, however, the bony plates behind the jaws have disappeared, leaving an exposed area, the otic notch, behind the upper jaw, where the sound-transmitting mechanism of the middle ear has already developed. Some of the bony plates on the jaws and all of the facial enamel have disappeared, but as yet there is no covering of soft, sensitive, flesh.

In the fossil, mammal-like reptiles, which represent the next stage of facial and dental evolution, the bony mask of the temporal region is perforated and the temporal fossa is foreshadowed. The thinning of the bone is possibly an effect of the absorbent action of the jaw muscles attached to its inner sur-

*THE SHARK

"The shark has teeth set like a saw
In serried rows within his jaw.
When one set gets the worse for wear,
He simply pushes in a spare.
With all of his successive dentures
He has carnivorous adventures,
And since he cannot bear the sight of you,
He rolls on his back to take a bite of you."

face. Below this opening is the forerunner of the mammalian zygomatic arch. Meanwhile, there is a tendency for the complex dentary (or lower jaw) to evolve from a mosaic of bony elements into a one-piece type (two halves connected at the front end or symphysis). First of all the dentary becomes enlarged and presses against one of the jaw muscles in which it is embedded (the external pterygoid) forming the cushion (meniscus) which in typical mammals prevents the lower jaw from grinding into its socket in the temporal bone. This same pressure somehow forms the glenoid fossa, in the temporal bone, so that a new mammalian joint is created between the dentary and the skull, whereas the old reptilian joint, which lay between the quadrate bone of the upper jaw and the articular bone of the lower, is reduced in size and pushed back into the ear to serve in the ear ossicles the new function of transmitting sound vibrations. The one-piece jaw is finally achieved with the evolution of mammals.

Heat-regulating devices were necessary for resistance of temperature changes which the evolving land mammals had to withstand. Among these were the diaphragm, the sebaceous glands, the sudoriferous or sweat glands, and the hair. It is possible that the hair, a heat-retaining structure and, at the same time, an insulator, grew out between the scales even in mammal-like reptiles. Certainly, it supplanted these scales in mammals, whereas the bony mask of the face was replaced by pliable skin underlaid by facial muscles. These facial muscles seem to have developed from a wide, thin band covering the neck and throat in reptiles, the primitive sphincter colli. In mammals it expanded into the platysma and sent out various branches. The forward migration and proliferation of an original neck muscle gave rise to the muscles that move the lips, eyes, ears, and express emotion—all, of course, at the expense of exposing tender and mobile soft parts to a hostile world addicted to "pushing in" faces.

The evolution of the teeth really outstrips that of the face, but the complicated processes of early differentiation from fish to mammal need not be detailed here. It may be remarked that odontologists have been more successful in devising a fearsome sesquepedalian terminology for the description of dental evolution than in elucidating its causes and defining its stages. It can be stated, however, that the tooth-bearing rolls of skin in sharks lie in depressions in the jaws and the teeth are tied into place only by dental ligaments attached to their bases. In the lobe-finned fishes the upper and lower jaws now include bone-bearing plates to which the teeth are fastened by a complex infolding of their bases. (Hence, odontologists call them "labyrinthodont"—after the Cretan labyrinth or maze, irrespective of the fact that *labrys* meant double "axe" in Cretan and the Labyrinth was the Hall of the Axes). By the time the mammal-like reptiles have evolved, the teeth are already set, each in its separate socket, as in man. In the earlier creatures these teeth were all alike, simple peg forms, more or less conical with a single point or cusp. These are retained in the canine teeth (or "cuspids"), but the other teeth are differentiated in form according to their position in the jaws and the function they subserve: the crowns of the anterior teeth expand to a chisel-form for biting

off food, the teeth behind the canines and nearer to the fulcra at the condyles of the lower jaw tending to become bicuspid, tricuspid, or quadrate with four elevated cutting points, for more efficient service in chopping and grinding. This differentiation of the original peg teeth into incisors, canines, premolars, and molars was foreshadowed in the mammal-like reptiles. Concurrently with this specialization in tooth form, the dentition was apparently reduced to two sets—a milk set and a permanent set, and reduction in the number of teeth proceeded to a point where the dental formula in Old World monkeys, apes, and man became

$$i\,\frac{2}{2}\quad c\,\frac{1}{1}\quad p\,\frac{2}{2}\quad m\,\frac{3}{3}$$

ARBOREAL FACES

The first primitive primates were evolved in the Eocene period, some sixty millions of years ago. They were tiny, long-snouted mammals, much like the tree shrews of today, but they were tree-dwellers (or arboreal), and the subsequent development of the higher primate face seems to be dependent upon this mode of life to a considerable extent. The primitive ground-dwelling mammal has to be provided with a long, pointed snout tipped by the nose, which is a principal organ of touch and the sole organ of smell. Since he cannot see above the long grass he is forced, as Wood Jones has said, to "nose his way through life," depending for the detection of food and enemies largely upon the tactile and olfactory functions of his soft damp muzzle, beneath which is conveniently situated the ingestive orifice or mouth, provided with some sort of teeth of naturally disposed in dental arches corresponding to the elongated shape of the snout. Now, when the ancestors of these primitive primates took to the trees, arboreal life effected a considerable diminution of the function of the face. In the first place, the primates were admirably equipped for tree life in the retention of a very primitive arrangement of hands and feet—five digits on each extremity provided with flat nails instead of claws and with the innermost digit on each—the thumb or the great toe—capable of being rotated so that the tip could be opposed to the rounded tactile bulbs of the outer digits. These prehensile hands and feet with their facility of tip opposition not only enabled the little animals to wrap their hands and feet around boughs or surfaces of any shape that their grasp could encompass, but also made it possible for them to pick up smaller and larger objects to be brought before their eyes for visual examination, to their noses for smelling, and to their mouths for eating. Hence the small primate up in the trees, by virtue of his manual equipment, tended to become in some measure a hand-feeder and manipulator, thus depriving the long, pointed, and primitive mammalian snout of its tactile and grazing function. However, it is clear enough that such a small arboreal animal would not have his forelimbs and hands free for exploratory and feeding purposes until to some extent they had become emancipated from locomotor function by a development of the habit of sitting up in the trees and sustaining the weight of the body mainly upon the hindlimbs. This tree-sitting, as we know, has been developed

in some rodents, such as the squirrel, and concomitantly, the habit of conveying food to the mouth with the paws, but the squirrels and other infraprimate arboreal mammals are handicapped by clawed digits that prevent any high development of the prehensile function of the hands. With the tarsier, a tiny, hopping primate found in the East Indies and a modern survival of an early progressive stage of primate evolution, sitting up and using the hands for exploring its own body and external objects, as well as for conveying them to the nose, to the mouth, and in front of the eyes, is closely related to the specialization of the hindlimbs for support and locomotion. This emancipation of the forelimbs (Wood Jones's term) is associated in the tarsier with a remarkable reduction in the length of the snout, with a tendency of the laterally placed eyes to swivel around to the front so that their fields of vision overlap, presaging stereoscopic sight; with a modification of the primitive, soft, damp muzzle or rhinarium into a nose tipped with skin instead of with mucous membrane. When atrophy of function takes place in an organ, it is axiomatic that the organ diminishes in size, and, with the shrinking back of the snout, the dental arcades were naturally shortened and became relatively broader behind. Thus the shape of the palate tends to be transformed from a very elongated V to a shorter, broader V.

In this connection it is important to realize that tree life puts a premium upon the sense of vision as contrasted with the olfactory sense upon which the small, weak ground-dweller has largely to depend for the maintenance of his lowly existence. Agility and motor coordination are required to permit an animal to move about the trees without falling; his eyesight supplants his sense of smell as the principal survival faculty. There can be little doubt that increased dependence upon vision tends to enlarge the visual areas of the brain at the expense of the primitive olfactory areas. So the brain expands, laterally and especially posteriorly in accordance with the location of the visual centers in the occipital lobes of the cerebrum, and, with concomitant shrinking back of the muzzle, the head tends to become nearly balanced upon the erect spine, facilitating the holding of it upright without the muscular effort caused by the vast overbalancing of the protrusive muzzle. Hence, we see set in train a chain of evolutionary consequences of upright posture in the trees and atrophy of snout function. The latter is accelerated not only by the substitution of visual for olfactory examination, but also by the fact that the animal no longer grazes, as the terrestrial quadruped is forced to do when all of his extremities are used for support and locomotion and he has to bite off his food with his teeth. This grazing function requires that the incisive tip of the jaws be considerably anterior to the eyes—to the visual plane—in order that the creature may eat and see at the same time—may keep his eyes out of his food. When the arboreal primate became a hand-feeder, the incisors and canines tended to become reduced in size, since food could be picked and prepared to some extent by the hands. The long, sharp, pointed canines, or eyeteeth, were ordinarily preserved, both for their defensive and aggresive function (since an animal without claws and with comparatively weak hands must resort to his teeth for combat). Furthermore,

in the trees the diet is necessarily largely fruit, nuts, shoots, and other tough vegetable material, for which long, sharp canine shears are of considerable utility, even when the traditional piercing and holding function of the carnivore's canine has fallen into abeyance.

The most primitive primates, lemurs and tarsiers, depend for survival upon the relative safety provided by their arboreal habitat, upon their agility, and upon their mainly nocturnal habits. The problem becomes more difficult when the animal gets larger and larger, as in the case of some monkeys and all of the giant anthropoid apes. The bulky tree-dwelling primate has to be careful about entrusting his weight to weak boughs; he cannot flip agilely from tree to tree, as does the featherweight gibbon. When he exhausts the food resources of a tree, the chimpanzee, which is about the body bulk of a smallish man, is forced to descend and travel along the ground. The gorilla, the weight of whom in adult males may exceed 500 pounds, rarely climbs trees after becoming full grown. The baboons, largest of the monkey family, are almost wholly terrestrial. These large primates, who usually adopt a quadrupedal gait on the ground, rely mainly upon their powerful jaws and tusklike canines for defense against beasts of prey. Hence, some secondary elongation of the snout seems to have taken place in most of them. In baboons and mandrills the muzzle has been extended to a most extreme and bestial degree and the canines are tremendously enlarged. Since this extension of the muzzle is accompanied by very little increase in the size of the brain and its covering bony envelope, the attachments of the muscles that operate the overbalancing jaws must be enlarged. The temporals cover nearly the entire cranial vault and in the adult males bony sagittal and transverse crests develop to afford additional purchase for the masticatory and supporting nuchal musculature. Further, in order to provide a buttress against the tremendous upward strains on the maxilla exerted during chewing, there develops in many of these large primates a torus or supraorbital bar of bone which sticks out like a shelf on the anterior border of the frontal. It is apparent, therefore, that in present-day large subhuman primates, who have adopted a partially terrestrial habitat, some retrogression toward lower mammalian types has taken place, perhaps in connection with a re-emphasizing of the defensive functions of the canine teeth and also because structural alterations are required when an animal's size is increased, in conformity with the law of cubes and squares.

However, there also does occur in the anthropoid apes a new cranial feature that reaches its fullest development only when humanity is attained. This is the bending down of the face upon the brain case. In the lower primates, such as the lemur, the craniofacial axis (a line extending from the anterior border of foramen magnum through the sella turcica of the sphenoid bone to the nasofrontal articulation) is straight and directed upward and forward. In the anthropoid apes and in man this craniofacial axis is bent at the sphenoid, so that its anterior leg is deflected toward the horizontal, while the posterior leg (more or less coincident with the basal process of the occipital bone) remains virtually unchanged. With the bending of the anterior half of the craniofacial axis, the whole face is depressed and the projection of the jaws diminished, while

the apparent vertical height of the face is increased. The animal now looks out from its nose, instead of along it. The radical change thus brought about in the relation of facial to cranial skeleton clearly conduces to the easier maintenance of the head poise upon the erect spine, since the weight arm of the lever is reduced by the bending down of the face. There is a possibility also that this bending of the craniofacial base actually has some retarding effect upon the forward or anterior growth of the face as influenced by the tremendous forces developed during the eruption of the molars. Dr. Lawrence Baker has shown, in yet unpublished researches, that in such a mammal as the pig the successive cheek teeth (molars) erupting downward from their crypts in the posterior part of the maxillary sinus form a species of curved lever with the application of the power anteriorly where the front edge of the erupting molar wedges against the last tooth already in position and the resistance furnished posteriorly by the pterygoid process of the sphenoid. Thus a mighty growth force, making for prognathism or protrusion of the jaws and of the whole face, is exerted between the facial skeleton and the craniofacial base, taking its origin, of course, in the downward and forward impetus of the erupting maxillary teeth. Correspondingly, in the mandible there is a wedging of the erupting molar between the posterior edge of the crown of the tooth already fully in place and the anterior edge of the tipped tooth in process of eruption. Again, in the mandible the resistance posteriorly is furnished for this thrust by the lower portion of the ascending ramus rising at a right angle or thereabouts to the crown-root axis of the erupting molar. The function of the lower part of the ascending ramus in the mandible is thus analogous to that of the pterygoid process in the maxillary eruption. Hence, the anterior growth of the face and jaws, which is so marked a feature of mammalian development at the time of the eruption of the permanent dentition is largely localized in the posterior parts of the maxilla and of the corpus of the mandible. With the sharp bending down of the face upon the craniofacial axis, there is apparently some inhibition of the eruptive growth force of the cheek teeth or possibly its direction is changed from a forward to a downward thrust, with consequent lessening of growth of the posterior parts of the alveolar borders. The result is decreased extension backward of the maxillary sinus during the latter part of tooth eruption, a similar decrease in growth of the posterior part of the corpus mandibuli, and ultimately a tendency toward molar impacting. Anteriorly, this evolutionary trend or functional alteration still further decreases the protrusion of the face already diminished in the course of its bending downward upon the craniofacial axis. Thus, there are complex and interrelated factors in higher primate evolution making for decreased prognathism and shortening of the dental arcades, and these processes, the etiology of which is obscure, have already begun at the evolutionary level of the giant apes.

THE FACES OF FOSSIL APES AND MAN'S PREHUMAN ANCESTORS

There is some disagreement among students of human evolution about the time when the human line branched off from the common trunk that leads to the

giant apes and to man. Some suppose that this separation occurred as far back as the Oligocene period (roughly 35 millions of years ago), when our prehuman ancestors were tiny ground apes. Others, with whom I agree, believe that this divergence did not take place until the Miocene period or later, when the gigantism had already affected the ancestors of the present great apes and our own stock. However, there is no doubt that our immediately prehuman ancestors were ground apes and possibly of size far exceeding that even of the contemporary huge gorilla. Recent theories of Weidenreich based upon discoveries of von Koenigswald in Java and South China include an apparently human mandibular fragment with the teeth in situ, both almost incredibly large *(Meganthropus palaeojavanicus)*. An even more gigantic precursor, according to Weidenreich, is represented by three teeth bought in a South Chinese apothecary shop by von Koenigswald and attributed by him to a giant ape (Gigantopithecus). This theory of giant human precursors should be held in abeyance until more evidence is available, if for no other reason because the distinction between fossil ape and prehuman molars, apart from associated skeletal remains, is a matter of opinion and guesswork, however vehemently the odontologist may argue and with whatever dental minutiae he may attempt to back his judgment.

We can assert, however, without much fear of dissent, that the unknown Pliocene ancestors of man had not yet attained a brain size greatly exceeding that of the modern gorilla (whose maximum cranial capacity seldom exceeds 600 c.c.), and that as yet there could have been little or no reduction in the facial prognathism and massive dentition that could be expected of a giant ape. This conclusion stems from the nature of the face-brain case relations in *Pithecanthropus erectus,* and *Sinanthropus pekinensis,* the earliest human fossils so far discovered, which date from the lower to the middle Pleistocene period (probably not more than 750,000 years ago), and in whom the cranial capacity ranges from about 750 to 1,000 c.c. as contrasted with the mean of 1,450 c.c., generally given for the adult male European specimen of *Homo sapiens.* Go back another million years and the brains of our prehuman ancestors could scarcely have exceeded greatly the figure given for out-size gorillas. Some small cerebral advantage over the ancestors of contemporary apes must have been enjoyed by the apes in the human lineage, but it may have been more in quality than in gross size.

I am belaboring this problem of the relation of size of brain to size of face and jaws in fossil primates because of the traditional belief that in some way the increase of mass of the former is causally related to the diminution of the latter in human evolution. I doubt that these two variables—size of brain and size of jaws—are really negatively correlated to any marked degree—such that the increase of one involves the decrease of the other. There may indeed be some rough physiologic compensatory interrelationship between size of brain case and size of face, in that only a certain amount of growth energy (blood supply) is available for the head and that an inordinate demand upon that limited supply exerted by the central nervous system might result in a diminished quantity to the jaws, or vice versa. "Robbing Peter to pay Paul" is an old habit within

animal organisms, whether acquired by imbalance of function or developed by genetic predisposition.

The best evidence bearing upon this question has been furnished in recent years by the discovery in South Africa of fossil man-apes (the Australopithecinae) in geological deposits of relatively late date, probably no earlier than Middle or Lower Pleistocene (from 500,000 to 750,000 years ago). The three fossil apes in question, discovered by Dart and Broom, are probably too late to be in the direct line of human descent, but they are far closer to man than any pithecoid form heretofore discovered. All of them dwelt in caves in an area of South Africa which is presumed to have been treeless, even back in the Pleistocene. They must therefore have been ground-dwellers, and the substantial portions of femora and other parts of the lower extremities discovered indicate unequivocally that they were erect in posture and biped in gait. Contrary to the case of other fossil ape discoveries (which have consisted usually only of teeth and jaw fragments), these South African fossils are represented by enough skeletal parts to enable us to judge of their status without odontological crystal-gazing. The teeth and jaws of these giant primates could be ascribed to a human animal with more credibility than to an ape. The incisors are rather small and not especially procumbent; the canines are reduced to the general level of the dental arches, with no interlocking and no diastemas or gaps between canines and incisors or premolars; the premolars are humanly bicuspid; the molars preserve the generalized fossil ape *(Dryopithecus)* pattern that is the common inheritance of gorillas, chimpanzees, orangutans, all fossil men, and undegenerate specimens of modern *Homo sapiens*. However, the molars are of huge dimensions and increase in size from the first to the third, contrary to the condition in modern man, in most fossil men (except *Pithecanthropus erectus*), and in the contemporary chimpanzee. The maxillas and mandibles, in which the teeth of these adult man-apes are implanted, are not markedly prognathous (in comparison with the most simian examples of fossil men). Yet, their morphology is distinctly that of apes. Furthermore, the brain cases, which are in a couple of instances well preserved, clearly show that the brains of these upright-walking small-jawed South African apes were only slightly, if at all, larger than those of present-day chimpanzees or orangutans, with whom they seem to compare closely in body bulk.

It follows that in the South African fossil apes (1) marked humanoid reduction of the dentition and decrease of prognathism occurred without any concomitant increase in brain size, and (2) erect posture and biped gait were achieved in association with diminished facial protrusion and reduced canines and incisors. It may be concluded that giant primates may undergo humanoid changes in facial and dental regression quite independently of brain growth, and that the erection posture may be a more potent factor in shrinking the jaws than any acceleration of brain growth, perhaps because an approximate balance of the head upon the erect spine is essential to the easy maintenance of the upright stance.

Let us consider also the bearing of brain case/face relationships upon this problem as revealed by the earliest specimens of fossil man. From Java are

now available sufficient fragmentary specimens of brain cases, faces, and jaws of *Pithecanthropus erectus* to permit a reconstruction of the skull of this erect-walking, very primitive man. In this type the brain case has increased in size so as to outdistance that of any anthropoid apes. Pithecanthropus cranial capacities vary from approximately 750 to 950 c.c. (roughly one and one-half times the capacity of the largest gorilla). Yet there is apparently no sign of facial or dental retrogression much beyond that shown by the South African, small-brained, short-faced fossil apes. Indeed, one specimen of Pithecanthropus (Weidenreich's *Pithecanthropus robustus*) has a maxilla in which is implanted a projecting canine separated from the lateral incisor by a distinct diastema. Further, in at least one of the Pithecanthropus specimens, the molars increase in size from front to back, showing that reduction in growth of the posterior parts of the jaws has not yet begun.

In *Eoanthropus* (the Piltdown skull), a brain case and half of the mandible discovered in England in an Early Pleistocene deposit (at least as old as any Pithecanthropus find), the incompatibility between evolutionary status and degree of development of the two main parts of the skull reaches its maximum. The extremely thick-walled brain case has a capacity of over 1,300 c.c. and is in nearly every respect morphologically modern. There is no low forehead, no great supraorbital torus, no anthropoid characteristic of the attachments of the nuchal muscles. Yet the associated mandible and a lower canine found near the brain case are almost wholly apelike. The canine projects, the jaw is chinless and provided with a simian shelf inside the symphysis; the molars in situ in the mandibular half might be either chimpanzee or human. While some authorities deny that the Piltdown mandible belongs with the skull, and attribute the former to some sort of fossil ape (an orang, according to Weidenreich), the consensus is that the association is correct and that *Eoanthropus* represents a precocious development of brain and brain case in size and morphology toward the *Homo sapiens* level, whereas the face and jaws are evolutionarily retarded and are mainly anthropoidal. This phenomenon of the association within the same organism of parts that are incompatible in that they represent different degrees of development from the primitive and anthropoidal is manifest in many early human fossils and the remains of the extinct apes. I have called it asymmetry in evolution. It suggests that a normal functional interrelationship of parts of the organism may be maintained when these have evolved at different rates and represent apparently incompatible evolutionary stages. It also means that in the early human and giant primate stocks, at any rate, growth of the brain and reduction of jaws and teeth are not closely interdependent and may be affected by diverse genetic and environmental factors.

In some of the later, but still rather simian types of fossil man, such as many of the Neanderthal specimens, there is more harmony between the evolutionary status of brain case, jaws, and postcranial parts. Yet extreme asymmetries within the same organism still manifest themselves. Thus, in most of the Mount Carmel specimens recently described by Keith and McCown, a mainly modern type of brain case has superadded a markedly simian supraorbital torus,

and the pronounced prognathism is mostly alveolar, as contrasted with that of the whole face; the chin eminence is well developed, and the postcranial skeleton is virtually indistinguishable from that of modern man *(Homo sapiens)*. It is my opinion that these Neanderthal features mixed up with those of *Homo sapiens* in the Mount Carmel specimens are the result of hybridization, but they can also be interpreted, as they are by Keith and McCown, to be rather due to the essentially asymmetrical character of human evolution. It is clear enough that individual inheritance may also show combinations of evolutionarily incompatible parts derived from the interbreeding of strains representing diverse evolutionary levels of development in specific features. It is equally certain that without such hybridization, some parts of the body may lag in an evolutionary sense and others advance precociously, perhaps as a result of differentiating environmental stimuli.

In early examples of fossil *Homo sapiens* (morphologically modern man) dating for the most part from the end of the Pleistocene—some 25,000 to 30,000 years ago—the brain has already evolved to its modern size, and often exceeds present averages; the face and jaws are robust, but not extraordinarily protrusive; prognathism is alveolar rather than total facial; the dental arcades are well developed, but the canines and incisors are reduced to more or less modern dimensions and the third molars are usually smaller than the second or the first. There is no sign of undue facial retrogression nor of dental degeneration, although it is clear enough that the evolutionary trend has already set in in an opposite direction from that manifested in, e.g., the gorilla and the orangutan. Dental occlusions are usually perfect.

THE GENESIS OF MALOCCLUSION AND DENTAL DISEASE

It is difficult to make any precise statement as to the period when the excessive shrinking back of the face, the constriction of the dental arches, and the phenomenon of dental malocclusion began to be prevalent in geologically recent man. The main races of mankind—Negroid, Mongoloid, and White—seem to have been affected to different degrees and at different periods by these accelerated retrogressive changes and they are associated more closely with the advance of so-called civilization than with the lapse of time. It may be stated that, on the whole, the White race has been most affected by these changes, the Mongoloids next, and the Negroids least. To some extent, this order corresponds with the probable antiquity and degree of differentiation into subtypes of these three main racial groups, with the exception of the fact that the Negroid subdivision is probably a later development than the Mongoloid. This ranking in facial and dental retrogression accords more closely with the varying extents to which these races have been subjected to the influences of "civilization" and the duration of time that they have been so exposed. Within any of the great racial stocks a subdivision that has remained isolated and has continued to live under relatively "primitive" conditions is likely to show diminished degenerative changes of the face, or, often, none at all. We know most about the Whites. In Egypt there seems to be comparatively little of these degenerative conditions

in evidence until a great civilization has developed in the Nile Valley—some time after 3000 B.C. While less skeletal evidence is available, it may be conjectured that in Mesopotamia and the Near East in general, marked facial retrogression accompanied by malocclusion follows upon the development of urban centers after 4000 B.C. In Italy, this phenomenon seems to begin with the rise of the Etruscan city-states and to manifest itself among the Romans toward the end of the Republic and the beginning of the Empire; roughly not much earlier than one century B.C. Further north and west in Europe there is usually little evidence of facial and dental retrogression (in comparison with the status of primitive *Homo sapiens*) until medieval times. On the whole, it seems probable that some reducing trend in facial and dental growth of long standing in evolution is suddenly accelerated and rendered definitely degenerative and pathologic when the White races come under the artificial regimes of city-dwelling and when they become dependent upon agriculture and especially upon cereal diets.

In the New World, where the aboriginal stocks are Mongoloid in varying degrees, the question of the origin and distribution of dental malocclusion is complicated by artificial cranial deformation which was widely practiced among the groups of higher cultural achievement. Frontal, fronto-occipital, or other distortion of the brain case naturally disturbs the hafting of the face and may well account for the malocclusions which are not infrequent. Here again, however, the incidence of malocclusions seems to coincide fairly closely with that of sedentary village life in fairly highly organized societies living on a mainly agricultural basis of subsistence.

The distribution of dental caries differs in some important respects from that of malocclusion, and, although the two are frequently rampant in the same stocks and the same individuals, they do not arise necessarily from the same causes. Dental caries occurs in a fair percentage of free-ranging, wild, higher primates, such as anthropoid apes, although much commoner in animals raised in captivity. It is a curious fact that decay of the teeth, except that which follows attrition and wearing down of the crowns until the pulp cavities are exposed and death of the tooth results, is almost unknown in fossil man and in hunting and food-collecting primitives right down to the present day. I know of but one specimen of apelike fossil man—the Rhodesian man—badly afflicted by caries, gingivitis, alveolar abscesses, and other dental diseases. In this specimen, which is of uncertain antiquity, but probably dates back to the Pleistocene, the mandible is missing. The maxillary arch is intact and, although of vast size, shows the horseshoe or elliptical form that results from reduction and rotation of the third molars. The teeth are so badly worn down that it is difficult to decide whether the extensive caries, pyorrhea, and alveolar abscesses with middle-ear involvements are consequences of the extreme attrition of an abrasive diet or whether the caries and periodontal infections preceded the wearing down of the teeth. Since many ancient and modern skulls show excessive wear of the teeth without accompanying pathology, it may be conjectured that Rhodesian man was the exception to the nearly total immunity to caries apparently enjoyed

by fossil man and by modern primitives. We, of course, know nothing of his diet or of his manner of living, but neither of these could have been refined or artificial.

Examples of comparative independence of malocclusion and dental caries may be noted in the skulls of ancient Hawaiians, many of which contain teeth of a pearly whiteness, absolutely free of decay, while impacted third molars and malocclusion are comparatively common. On the other hand, Weston Price has adduced many instances of primitive peoples observed by him, among whom the older generations have perfect occlusions and freedom from dental caries and other diseases, whereas their children who have been brought up on canned food, white bread, sugar, and other imports from the civilized world, present all sorts of defective developments of the dental arches and rampant caries.

THE CAUSES OF MODERN FACIAL RETROGRESSION

It is important for the orthodontist to decide whether in attempting to correct malocclusion he is combatting an evolutionary trend, possibly of an orthogenetic nature, or whether he is attempting to remedy a pathologic condition that is the result of infection, of atrophy of function of the masticatory apparatus arising from dependence upon soft cooked food, of subnormal bone development due to deficiency diets, or of all of these factors in combination.

On the evolutionary side, it should be noted that animal organisms are prone to get into evolutionary ruts whereby the development of a bodily part proceeds to the extreme in one direction or another until checked by natural selection through the elimination or extinction of the animal possessing exaggerated variations that are unfavorable. Inbreeding is conducive to such a piling up and accentuation of defects. In the famous descendants of the Mutineers of the Bounty and the Polynesian women on Norfolk and Pitcairn Islands, H. L. Shapiro found that bodily health and physical development were in general excellent, but that, almost without exception, practically all teeth were lost before maturity. He was not able to ascertain that this premature dental loss could be attributed to the devastation of caries. The Pitcairners merely said that their teeth were not good, gave trouble, and that they had them out. There is no particular indication that dietary deficiencies are responsible for this Pitcairn situation. It seems more probable that in this population, inbred for some seven generations and descended from only a few British sailors, a genetic tendency toward poor teeth has been broadcast and accentuated by inbreeding. Many of the British ethnic types exhibit facial reduction and malocclusion in extreme degrees, in addition to great susceptibility to dental caries. In the Pitcairners, if I am correct, the liability to dental caries or generally soft and inferior teeth has been inherited. It is difficult to say what the occlusive status of these people may have been, since they have practically no teeth at all in adult years. We observe many cases in our own population in which it is obvious that defective development of the face in parents is paralleled and exaggerated in their children. It is hard to see how these conditions could be duplicated, except through heredity, especially in modern families in which the

children are carefully reared from infancy on diets prescribed by orthopedists and modern nutritionists, whereas their parents for the most part were like Topsy in that they "simply growed" without benefit of cod-liver oil, capsulated vitamins, spinach, orange juice, and the rest of it.

On the other hand, instances have come under observation of all of us, in which parents with satisfactory occlusions produce children in which the relationships of dental arches and teeth have gone sadly awry in spite of careful attention to the diet and care of the teeth from early childhood. It is easier to explain the appearance of such malocclusions by invoking the doubling up in the offspring of recessive factors making for dental malocclusion transmitted by the parents but not visible in them, than by resorting to environmental apologies. I think that we ought to face the probability (for I do not know that it has been demonstrated as a statistical fact) that serious disturbances in the growth and development of the face are increasing alarmingly in recent generations in spite of putatively better infant care and feeding and a generally improved nutritional status.

There is another possibility of genetic causation of the proliferation of malocclusions in our recent crops of children and young people. Increased transportation facilities, the influx of immigrant peoples representing widely varied physical ethnic types, and the generally greater mobility of our American population have resulted in far more out-marrying between individuals of widely diverse facial and cranial types than ever occurred in the past. It may be that such outbreeding has produced in some of the offspring disharmonic combinations of teeth, jaws, and brain cases that are manifest in dental malocclusions. It seems almost certain that some of the disturbances in dental relationships adduced by Weston Price in the children of primitive peoples are attributable to hybridization. However, I should doubt that genetic disharmonies or dysplasias due to outbreeding are a major cause of our present occlusal predicaments. The matter cannot be settled one way or the other without elaborate investigations of the inheritance of facial types and occlusions.

CAN RETROGRESSION OF THE FACE AND TEETH BE CHECKED?

What we know about the etiology of malocclusion (which is all too little) can now be summarized before we ask ourselves what can be done about the problem. In the first place, it is all too evident that in the White race and possibly in Mongoloids and Negroids there is a long-standing and deep-rooted evolutionary trend toward both reduction of the bony structure of the face and diminution in size of the dental arcades and of the teeth themselves. This trend manifests itself in different ways and to different degrees in various racial and subracial types. Broad-faced, short-faced, round-headed peoples, for example, are much more likely to suffer from impacted molars due to insufficient posterior development of the jaws than are long-faced, narrow-faced, long-headed peoples. These latter are prone to malocclusions involving insufficient width of the palate and mandible, defective or arrested mandibular development, as contrasted, perhaps, with lack of the anteroposterior growth of the middle face in

roundheads. However we look at it, we are up against retrogressive factors that have got into the germ plasm and cannot be eliminated except by selective breeding in so far as they are hereditary. Bearing this unpleasant fact in mind, it is perhaps more practicable to attempt merely to restore function within the various patterns set by heredity than to oppose the whole evolutionary trend and to try to reshape the face in an ancestral mold.

There can be no doubt that in some measure modern malocclusion is encouraged, if not actually caused, by defective and arrested growth referable to improper diets, pathologic conditions in the nose and throat, or generally poor health that affects the entire organism. It is then clear that mechanical correction of malocclusions in a person whose whole physiology is subnormal or disturbed can be of little use. The orthodontist who moves teeth and reshapes dental arches has to assume that a healthy organism will take over and maintain in functional equilibrium the finished product of his scientific alterations. There is no use in attempting to reverse an evolutionary trend in a deteriorated, sickly, and malfunctioning organism. First of all, generally good physiologic function must be restored. It is hard enough for a swimmer to make headway against a strong current when he is in the pink of condition; if he is weak from sickness, it is impossible.

In the functional atrophy and shrinkage of an organ, whether due to hereditary or environmental causes, the different parts of that organ are probably affected in the order of their comparative plasticity. Thus, in the masticatory apparatus the muscles are first decreased, then the bone, and finally the teeth. This is probably the explanation of the fact that the jaws are not big enough for the teeth to erupt in their proper positions. It is easy enough for the orthodontist to move teeth, but they must have an adequate bony setting—a firm foundation—if they are to stay put. The extent to which mere readjustment of the occlusal relations stimulates reaction in new bone growth to stabilize the teeth in altered positions is doubtful. In many cases it probably does not take place at all. Hence I should regard it as probably a sounder practice in orthodontics to remove a few teeth in order to make room in an inadequate dental arcade for the proper placing of the remainder than to attempt to warp all of them into a position that cannot be maintained because they have nothing to stand upon.

In conclusion, I should say that to me, man appears to be an animal that has entered upon a terminal and decidedly retrogressive evolutionary course as respects not only his teeth, jaws, and face, but also his brain case, its contents, and many other body parts. Brains, as well as jaws, seem to be shrinking. This general evolutionary decline is certainly attributable in part to functional atrophy—to the fact that man has tried to substitute the use of machines and gadgets for the proper exercising and performance of work by his own organism. He uses millstones for the grinding of his cereal food, choppers for the trituration of his animal food, chemists for the production of food that requires no chewing whatsoever, and then wonders why his face, which is mostly a setting for his masticatory apparatus, shrivels and warps. He cuts off the tails of a few

generations of unhappy mice and, because this mutilation is not inherited, jumps to the conclusion that atrophy of function in parents cannot affect the development of those disused parts in offspring, without pausing to survey the infinity of palaeontological evidence of gradual, then accelerated shrinking, and final disappearance of such obsolescent body organs, irrespective of their advantage in the struggle for survival. He patches up and preserves deteriorated human organisms and by the bounty of charity encourages them to proliferate and perpetuate their germinal defects and their sheer evolutionary pauperism. He tries to get along in the world without mental or physical effort and if he is capable of such healthful exertion, expends it all too often in stupidly helping the survival of those who ought not to survive, thus making a bad evolutionary matter much worse. Let the orthodontist contemplate the Biblical adage: "The fathers have eaten a sour grape, and the children's teeth are set on edge." This may not be good biology, according to Weismann, but it should offer some solace to the dental scientist who is bravely attempting to reverse the course of human evolution. If he fails, it at any rate provides him with an alibi.

ANTHROPOMETRY AND ORTHODONTICS

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A PURE or theoretical science undertakes by experimentation and deduction to discover principles, laws, causes, and effects pertaining to different classes of phenomena. Presumably, the purpose of most theoretical sciences is to accumulate knowledge that ultimately may be applied, in some way or other, for better rather than for worse. However, a great deal of the science that is pursued in academic confines is quite dissociated from application and even from aspirations of usefulness. It is carried on by self-sequestered individuals who probably have never asked themselves of what use to the world their work is, and some of whom glory in their remoteness from all human endeavor that can be called "utilitarian" or "gainful." Theirs is science for science's sake—"pure" in that it is purely useless.

Now, rightly or wrongly, I have become "fed up" with the kind of science that remains theoretical merely because its devotees regard themselves as intellectually and spiritually superior to those who apply knowledge in order to produce something the world needs. Science ought to be socially responsible even when not economically self-supporting; it should envisage its aims with reference to human society and should foresee the nature of the applications of its discoveries to man. It is partly lack of foresight and responsibility of "pure" physicists that has got the world into the atomic bomb mess.

Applied science might be considered a child of theoretical or pure science that puts to work the laws, principles, and techniques discovered by its parent. Actually, most applied sciences arise from the necessity of fulfilling immediate and urgent human needs, and the practitioner goes to work upon the problem by trial and error, without utilizing the antecedent results of a theoretical science, because the latter does not exist. The pure science and the theoretical knowledge are by-products of their application and the child is father to the man. So, physical anthropology is in large measure a theoretical by-product of the applied sciences of human anatomy and medicine.

In the past, physical anthropology has concerned itself mostly with the attempt to learn how and when and by what stages man evolved from lower animal forms, and, as regards contemporary man, his classification into great groups of individuals possessing similar hereditary physical combinations—races. Anthropologic studies have been, for the most part, historical, descriptive, nonfunctional, and dissociated from any sort of therapeutic application. True, anthropology has attempted to deduce general principles of evolution

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as well as to plot the actual course it has taken; within the human organism it has tried to study the interrelationship of parts and the range of their individual and racial variation. It has usually refused to consider the individual except as the unit of which human groups are composed. Following this tradition, I used formerly to disclaim as an anthropologist any interest in the individual human being, asserting that it was the business of the science I practiced only to define large human groups and to describe their attributes. I now regard such an attitude as undefensible and downright stupid, because group characteristics are only abstractions of a statistical nature, which may indeed lead to the formulation of principles and generalizations, but have little reality in a world of unit human organisms. You cannot improve races as such; you cannot cure populations of disease; there is no real therapy and no valid diagnosis unless applied to the individual man.

Now, orthodontics is an applied science. It has all of the strength and weakness of a system of procedures that has arisen out of the necessity of doing something immediately about a bad biologic situation, without waiting to investigate its genesis in the Garden of Eden or among our ape ancestors, and without considering such recondite biologic formulations as arise from the application of Mendel's laws of heredity to the breeding of fruit flies. The orthodontist was originally a plain dentist who saw that something had to be done with the human dentition besides filling and extracting teeth and fabricating dentures. Beginning, perhaps, with the crude fact that some teeth "needed straightening," as the layman puts it, the orthodontist rapidly proceeded to the knowledge that man's face and perhaps his whole organism needs straightening, and that wires, wedges, and other devices for moving teeth constitute a wholly inadequate apparatus for such an extensive biologic rehabilitation. Now, faced with the discouraging fact that he should bring to his task not only the mechanical skill of the dental specialty, but also an expert knowledge of nearly every item of human biologic science, the orthodontist turns hopefully for assistance to the various specialists in the apposite fields of science. Of the geneticist he asks information about the manner of inheritance of facial and dental abnormalities. He finds out that the geneticist knows only how to produce a Drosophila with pink eyes and a gray body and vestigial wings, or what rate of mutation may be expected to occur in the evening primrose. So, he goes on to the physiologist and asks him the relationship between hypothyroidism and the shape of the maxillary arch. If our orthodontic inquirer is lucky, he may come away from the physiologist with some information about the effect of iodine-deficient diets upon rats. Probably he then resorts in desperation to the anthropologist with an inquiry about the normal relationship of implantation of the maxillary first molar to the jugal crest and discovers that the cheerful specialist in human morphology is entirely preoccupied with the epicanthic fold of the upper eyelid or the effect on intelligence of cradle-board flattening of the occiput in prehistoric American Indians. Thus the orthodontist, a practical scientist, is frustrated in his attempt to get help from theoretical or pure scientists. He asks for bread and they give him a stone.

We may reasonably inquire how anthropometry *might* assist orthodontics and then examine the extent to which pertinent anthropometric data are now available for that purpose. First, however, I may offer the excuse that professional physical anthropologists are far fewer in number even than dentists who take only orthodontic cases. A large proportion of the small membership of the American Association of Physical Anthropologists are teachers of human anatomy in medical schools, geneticists, medical or dental practitioners, and even palaeontologists. Those who really profess physical anthropology usually have to teach other branches of anthropology also, such as primitive sociology, archaeology, etc. Only two or three positions in the United States allow the incumbent to devote himself solely to the teaching of physical anthropology and research in that subject. It should be understood, then, that the data of physical anthropology are meager partly because opportunities for making a living in this subject are few.

Anthropometry should assist the orthodontist first of all by providing him with the evolutionary background of malocclusion. Second, it should help determine the genetics of malocclusion and the ecological conditions associated with it. Third, anthropometry ought to provide data showing the range of normal morphologic variation in man, especially as concerns the jaws and the teeth. Finally, it might be expected that the physical anthropologist would be active in establishing precise relationships between the position of various teeth and of other facial parts during growth and after maturity. Let us see how far these orthodontic desiderata have been furnished by physical anthropology.

EVOLUTIONARY BACKGROUND OF MALOCCLUSION

Possibly the anthropologist has been most nearly a satisfactory collaborator of the orthodontist in establishing the general trends of human evolution that have led the modern status of man's masticatory apparatus. However, he has been hampered in this task by the fragmentary character of the skeletal remains of fossil man and because collections of crania of recent man, savage or civilized, rarely include a large proportion of specimens with all of the teeth in situ. Most skulls in museums lack some or all of the single-rooted teeth, which have been lost in life or subsequently. Aged specimens with the alveolar processes absorbed are of little value for dental studies. Children's skulls are nearly always disarticulated, warped, and incomplete. They can be reconstructed, if at all, only by inordinate labor. Yet, the greatest obstacle in the path of the anthropologist who tries to trace the evolutionary background of malocclusion is his comparative or complete ignorance of the classifications and problems of orthodontics. He needs instruction from the orthodontist and a clear statement of the latter's requirements of him.

GENETICS AND ECOLOGY

In spite of the large number of geneticists engaged in teaching at universities and in carrying on research there and in agricultural experiment stations, human inheritance is one of the most neglected fields of man's biology.

The excuse usually given for this disgraceful situation is the length of the human life span which makes it impossible for anyone except a Methuselah to carry on observations over a number of generations and the impracticability of controlling human subjects in their reproductive activities. These are indeed serious difficulties, but it is perfectly feasible to get the desired data by observing parents and children and even siblings. A more potent reason for the neglect of human heredity is that geneticists are afraid to study it. Among social environmentalists and uplifters there is a tremendous prejudice against the influence of heredity, easily propagated and fostered in the mass of the people who are afraid of their personal inheritance or ashamed of it. This prejudice has been worked into politics and insinuated into social policies, so that any reference to the importance of heredity or any projects for the study of human heredity—let alone public measures for the improvement of human heredity—are considered by the majority of our population to be undemocratic or downright fascistic. The situation has been aggravated, of course, by the employment of pseudoeugenics and racial theories as weapons whereby Hitler and his gang eliminated in Germany and elsewhere the minorities supposed to be unfavorable to the Nazi regime. Thus respectable studies and applications of genetics have tended to be carried on mainly for the benefit of the agriculturalist and the animal breeder.

Nevertheless, the apathy toward the study of human inheritance in medical schools is inexcusable. As far as I am aware, human genetics is not regularly a part of medical curricula and I doubt that human geneticists are to be found in many medical schools or institutions given over to medical research as permanent staff members. As long as medicine persists in its almost exclusive interest in bacteriologic and nutritional causes of disease and in treatment and cure, and ignores the constitutions of the animals that contract diseases, the essential facts of human heredity will remain unknown. The medical profession alone has full access to man for the study of his biology and it is up to the doctors to do something about it.

Now, if the orthodontists wish to learn about the inheritance of malocclusions, they will have to set up their own research projects and make their own observations on parents and children. Each practitioner ought to make it a part of his routine to take casts of the occlusions of the parents of his patients before going to work on the offspring, and, if possible, to secure dental casts of the siblings. Although it would be impracticable to study the whole family whenever any one member comes into the office with a toothache, it is likely that most parents who can afford to have their children receive orthodontic care over a period of years could be induced to put in an hour or two themselves furnishing the genetic background of the case. Further, in the orthodontic clinics of our dental schools and in institutions like the Forsyth Dental Infirmary of Boston, research projects for the study of occlusal inheritance should be initiated, while at the same time growth and nutritional studies are being made on the same patients.

There is no way of discovering the ecologic factors that are involved in the causation of malocclusion except by investigating the health, diet, and general environmental circumstances of persons showing such growth disturbances and at the same time of those free from the same defects. If it is impracticable to establish norms by the study of children with satisfactory occlusions, a control group of patients who are given satisfactory diets in the clinic should be set up. Anyhow, such a measure would probably be necessary for the correction of deficiencies in facial growth in that part of the population which depends upon free clinics for medical and dental care.

RANGE OF THE NORMAL DURING GROWTH AND IN THE ADULT

The physical anthropologist has set for himself the task of determining the range of normal variation in man and of classifying such variations. In so doing he has accepted a much larger order than he can fill. Consequently, the anthropologist has habitually limited his studies by excluding those specimens that are patently pathologic and concerning himself only with the range of variation within the putatively "normal." This unfortunate circumstance has been due in part also to the fact that most physical anthropologists are not medically trained, or, if they have been, they are still incompetent to determine the boundary between normality and abnormality in most pathologies, since the transitions are often so gradual that they call for the diagnosis of a specialist in whatever type of disease or defect is under investigation. Some years ago I listened to an argument at a dental convention between Dr. Aleš Hrdlička, the veteran and famous physical anthropologist (who began his career in medicine) and Dr. Kurt Thoma of Harvard University, the oral surgeon. The point at issue was the nature of the so-called mandibular and palatine tori—whether they were pathologic, or, as Dr. Hrdlička asserted, normal functional responses to excessive use of masticatory apparatus. Each disputant was sure that the other was wrong, and perhaps both were right, but the argument got nowhere except to convince the auditors that the correct solution of the problem was still to seek.

The average anthropologist who undertakes to study the range of morphologic variation is likely to pick out some feature that is of purely taxonomic or classificatory significance, such as the form of the articulation of the frontal, sphenoid, temporal, and parietal bones in the so-called pterion region under the temporal muscle. He selects such a functionally unimportant detail partly because he wants to avoid variations that are affected by specific pathologies which will disturb hereditary transmission and spoil the feature as a hallmark of race or of some other group classification. Such limitations in the exploration of the "normal" render the study simpler for the anthropologist but practically useless to the clinician.

Again, the anthropologist who is lucky enough to have his laboratory in a museum usually has to depend in his study of normal variation of skeletal parts upon material dug up by archaeologists, which lacks any accompanying information as to age, sex, medical history, nutritional status during life, and personal

identification. Unknown savages and civilized men, ancient or modern, whose bones happen to have been preserved and dug up, furnish only the means of ascertaining the range of variation within different ethnic and racial groups and between them. Rough indications of age changes can usually be made out. Family relationships between skeletons excavated from the same site, are of course, unknown, and so are usually the causes of death unless due to serious trauma or to disease that affects the bones. Almost the only exceptions to the anonymous and unidentifiable character of museum skeletal series are those collected at Western Reserve University by the late T. Wingate Todd, and at Washington University by R. J. Terry, now retired. These two large series of identified skeletons with medical histories have been studied, for the most part, only for age changes and for the distribution of a few anatomic features. They are a veritable treasury for the exploitation of the anthropologic research worker and may be of great potential value to the orthodontic investigator.

Physical anthropologists are now beginning to turn away from the racial classification of living groups and from skeletal studies oriented toward the mere description of morphologic and metric central tendencies. Many of us are now concerned rather with the morphologic, metric, and physiologic correlates inside the unit human organism, with the relationship of these to temperament, to psychology, and to social capacity. In order to obtain valid generalizations from such studies large series must be examined, but interest remains concentrated in the individual rather than in the group as such. The derived generalizations are of principal use in their application to single, living persons. These studies of the human constitution must usually be carried out on living persons, because the physiologic, pathologic, and sociologic associations of each specimen in a skeletal series are nearly always unknown.

A workable system of classifying body build into some 80 types has been devised by William H. Sheldon and is now being applied with great success. However, as yet we have very little specific knowledge about relationships of different forms of the dental arch, of different classes of malocclusion to these modern classifications of body build. Such a knowledge can readily be acquired if an orthodontic examination of subjects is included in the somatotype technique, which consists, as far as actual observations on the patients are concerned, of only a very few bodily measurements and three carefully posed photographs in the nude. There is little doubt that a collaborative study by orthodontists and constitutional anthropologists would establish important relationships between body type and form of face, jaws, and occlusal status.

Studies of the growth of infants and children are not at all new. Certain research institutions have devoted most of their time to such investigations for decades. Usually they have been almost totally sterile in their yield of utilizable results. Mostly, the data produced have consisted of growth curves for different body dimensions, means or "standards" of height, weight, etc., for children of different ages, derived from racially heterogeneous series and accompanied by inadequate information on the health, nutritional status, etc., of the individuals who comprise the series. No attention has been paid to the

different body types of the children studied. Much, then, of this growth study has been labor in vain. About all that has been found out about the growth of children is that they do indeed grow on the average so much, in such and such a measurement, between this and that age. A good deal of this work has been performed by educational psychologists or other classes of workers who have little comprehension of the problems of human biology and only the slightest acquaintance with modern technique of human measurement.

Of course there are some notable exceptions to the futility of conventional growth studies. The Brush Foundation at Cleveland has carried on over a period of time growth studies of children oriented toward the relationship of body development to health and to nutrition. To us here, the most important aspect of the work of the Brush Foundation is the participation in it of Dr. Holly Broadbent, who, by the use of his beautiful and accurate apparatus has carefully mapped the growth of the face, the dental arches, and the relationships of individual teeth. This at once brings to mind also the equally splendid equipment for the accurate determination of dental and facial relationships during growth and at maturity devised by Herbert Margolis. It is most regrettable that the Margolis apparatus has not yet been used in any extensive growth study.

This leads me to the climax of my discussion, which is, simply stated, that the techniques and precision instruments of Broadbent and Margolis, both orthodontists, in my opinion, have already contributed more to scientific knowledge of the growth of the face and of the teeth, and of the interrelationships of individual parts in this region, than have all the anthropologists and other growth workers with their primitive caliper measurements and their statistical manipulations of vast series of raw and inaccurate measurements. I believe that the solution of your problems and those of other specialties concerned with growth and the precise relationships of anatomical features in health and in disease lies in the widespread utilization of the Broadbent and/or the Margolis apparatus (for both are admirable) by skilled members of your profession working in collaboration with anthropologists studying the rest of the body and with medical investigators. It is my belief, further, that the orthodontic profession, whose leaders already know much more about facial growth and dental development than do anthropologists, pediatricians, anatomists, or other workers who have been more commonly engaged in this field of study, should assume responsibility for its planning and its execution. Orthodontics is a problem of total growth and development, not restricted to the part of the human organism that ordinarily engages your attention.

Here are some of the problems of orthodontics to which anthropometry, whether practiced by anthropologists or by orthodontists, can furnish the answer:

1. *Shape of the Dental Arches and Their Relation to Face Form and to Head Form.*—Anthropologists have roughly classified the maxillary arch as U-shaped or hypsiloid, parabolic, hyperbolic, and elliptical or horseshoe-shaped. Within each of these categories a wide variation is possible. Although it is known that the narrower, U-shaped forms are likely to go with narrower faces, and broader, hyperbolic forms with broad faces, that is about as far as the correlation has

been carried. Little consideration has been given to the shape of the mandibular arch relative to that of the maxillary arch. Long aware of the variation in shape of the dental arches within the limits of normal occlusion, anthropologists, of course, offered objections to arbitrary schemes of "predetermination" made on the basis of engineering devices for plotting the curves of the arch. Such techniques seem to assume the existence of but one so-called "normal" form of arch for whatever shape of head and face.

2. *The Etiology and Mechanisms of Dental and Facial Growth and Development.*—In this field of investigation, the anthropologist has hardly got beyond repeated measurement of the face throughout the period of growth, calculating the percentage relationships of one dimension to another, and recording the approximate times of eruption of the different teeth. Such crude data are of little value to your specialty. Sir Arthur Keith, indeed, has speculated brilliantly upon the causes and processes of facial growth, bringing to bear upon the subject his vast knowledge of the subhuman primates and of fossil man. It remained for Lawrence Baker, an orthodontist, to revive a method, originated, I think, by John Hunter, whereby madder is fed to experimental animals in order to study the amount and the location of bone growth under various circumstances. Dr. Baker's experiments in which he removed the tooth bud unilaterally and then studied the growth symmetries resulting from such mutilations are familiar to you. The experimental morphologic approach is difficult, expensive, and time consuming, but it is rewarding. Since experiments upon human subjects are impracticable, it is indicated that a much wider use of laboratory animals should be made in the future if the orthodontist is to understand the physiology of growth as it pertains to face, jaws, and teeth. The whole problem of the successful anchoring of teeth moved into normal occlusal relations depends upon the acquisition of such knowledge.

3. *The Norms of Skull Proportions and of Dental Relations.*—Planes of orientation of the skull and of the head, as devised by anthropologists, usually for study of skulls only, are crude and ill-established. They predate x-ray photographs of the skull, and I am not prepared to defend them as anything else than temporary expedients that will serve only until they are supplanted by accurate observations made by such apparatus as that contrived by Margolis and Broadbent. The important subject of the measurement of cranial and facial asymmetries relative to various planes has never been successfully developed by anthropologists because they have had no instruments adequate for the precise plotting of anatomical points on a three-dimensional scale. I may remark, parenthetically, that the practitioner of physical anthropology is likely to be mechanically ungifted. He has not the extraordinary engineering ability that is a commonplace among orthodontists and possibly a virtual prerequisite for the practice of your specialty.

THE NEED FOR COLLABORATION IN ANTHROPOLOGIC AND DENTAL RESEARCH

All of the unhappy remarks I have made about anthropometry and orthodontics point to the conclusions that a few overworked orthodontic practitioners,

however brilliant and skilled, and still fewer anthropologists, impractically oriented and unskilled in the application of their knowledge, are unlikely to lick the vast evolutionary and pathologic problem of malocclusion. To cope with it successfully we need properly endowed institutes for the study of human growth with special reference to dental development. The enlightened and progressive members of your profession should organize for the establishment of such an institution. I am afraid that most of the dental schools are too subordinate to other branches of medicine, too much submerged in curricular difficulties and in trying to compete with medicine for the enlistment of superior students, to achieve such a task. Perhaps orthodontists as a body should embark upon this project independently. I am confident that a reasonably endowed institution for orthodontic research would have no difficulty in recruiting for its staff competent young physical anthropologists and other scientists whose researches are essential to the solving of orthodontic problems. They will come running if assured of ample research opportunities and a reasonable living wage.

I doubt that unsurmountable difficulties would be encountered in raising funds for such a purpose from medical and charitable foundations. Some support might be expected from private donations within your own specialty. I am well aware of the extent to which many of you pour your own earnings into your private research. If these gifts could be channeled into the work of an organized institution with a full-time research staff to collaborate with individual experimenters, the latter would get more for their money and for their expenditure of time. Orthodontics would then achieve the scientific security and the high repute that it deserves.

THE REACTION OF THE PULP TO PRESSURE

M. B. Markus, D.D.S.,* Philadelphia, Pa.

FROM time to time, investigators have reported their histologic findings upon teeth and their supporting structures which have been subjected to pressure. The pressure employed was usually derived from some type of orthodontic appliance.

Their observations of changes occurring within the pulp of teeth subjected to pressure vary considerably. Some contend that practically no change takes place, others claim that only slight changes usually of a temporary character occur, while still others demonstrate definite degenerative changes within this dental structure. Some authors state that if destructive changes do occur within the pulp, the tooth has probably been subjected to trauma prior to orthodontic treatment.

It is therefore apparent that from an histologic standpoint there is no agreement as to the nature of pulpal changes after a tooth has been subjected to orthodontic treatment, and, if changes do occur, whether they are of a temporary or permanent character.

From a clinical standpoint the question of pulpal changes resulting from orthodontic treatment has received little attention. Many orthodontists agree that pulp devitalization may follow the application of severe pressure. One orthodontist has reported that as the result of changing his principles of treatment no devitalizations have occurred in his practice. Prior to this change in procedure he had observed eleven of such mishaps, and, with one exception, all had a history of trauma. It is quite possible that some teeth which have been traumatized may remain in a fairly healthy state but, if they are subjected to orthodontic treatment, degenerative changes may develop within the pulp.

Inasmuch as everyone is aware of the seriousness of pulpal involvement it should be of value to the orthodontist (1) to know whether orthodontic treatment has any effect upon the pulp and (2) to determine the state of health of the pulps of teeth prior to treatment, particularly those of the upper four incisor teeth, as they are the ones in children usually subjected to injury. Should pulps of questionable vitality be detected prior to treatment it would serve as a signal to the orthodontist to exercise extreme caution. The patient could then be apprised of the probabilities of future pulpal pathology.

Many times, radiographs do not reveal distinct changes which can be detected by the operator, which would inform him of the status of the pulp.

Presented before the New York Society of Orthodontists, March 4, 1946.

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Fig. 1 is a photograph of a boy 14 years of age. The color, shade, and translucency of the central incisors appear normal. Fig. 2 is the radiograph of the upper incisor teeth of the same patient taken at the same time. There is very little radiographic evidence that would lead one to question the state of health of the pulps of these teeth. Two years prior to this time the patient received an injury causing marked mobility of the upper left central incisor, resulting in considerable displacement of this tooth from its original position. Recalling this incident, further examination was decided upon, which revealed marked pulpal changes within the upper left central incisor. Had the author not recalled the previous injury which prompted detailed examination, the orthodontic treatment might be held responsible for injuring the pulp of the left central incisor tooth.



Fig. 1.



Fig. 2.

To obtain further information on changes that may occur within the pulp as the result of pressure created by an orthodontic appliance, it was decided to employ the electric pulp-tester. The value of testing pulps by means of the electric current is a rather controversial problem, especially as regards accuracy, type of apparatus, and technique employed. Ziskin has advocated the testing of

pulps at the threshold of stimulation by the measurement of the amount of current used as well as the voltage, laying particular stress on the amount of current.

After considerable experimentation, an electronic pulp-tester was constructed, employing electrodes that offered considerable stability. This was a highly sensitive instrument and enabled the operator to duplicate previous readings very closely, which was of extreme importance in carrying out this study.

The upper right and left central incisors of twenty-seven children varying in age between 11 and 14 years, making a total of fifty-four teeth, were tested. One of the teeth was nonvital and therefore was not included in this series. The testings were made several times to check for accuracy of reading. They were then ligated to an 0.030 round labial arch by means of an 0.010 stainless steel ligature wire in the manner usually employed when this type of appliance is used. The teeth were again tested immediately following the application of pressure supplied through the medium of the tightened ligature. These latter registrations revealed that in fifty-one of the fifty-three cases the readings were relatively different from those taken prior to the application of pressure.

An analysis of the readings taken following the application of pressure are shown in Table I. It can be seen that in sixteen teeth, or 30.1 per cent of the cases, there was an increase in the amperage with no appreciable change in the voltage. In fourteen teeth, or 26.5 per cent of the cases, there was an increase in amperage associated with a drop in voltage after pressure was applied to the teeth. In fifteen teeth, or 28.3 per cent of the cases examined, there was no appreciable change in amperage while the voltage dropped. In six teeth, or 11.3 per cent of the series, there was a drop in amperage as well as voltage following the application of pressure. In this group the voltage showed a proportionately greater drop than the amperage. In the remaining two teeth, or 3.8 per cent of the cases studied, there was no appreciable change in the pulp-test

TABLE I. ANALYSIS OF PULP-TEST READINGS

53 CASES	AV +o	AV +−	AV o−	AV −−	AV oo
Number	16	14	15	6	2
Per cent	30.1	26.5	28.3	11.3	3.8

A = Amperage recorded in microamperes.
V = Voltage recorded in units of voltage.
+ = Increase.
− = Decrease.
o = No change.

TABLE II. RANGE OF READINGS ON 53 CASES

	MICROAMPERES		UNITS OF VOLTAGE	
	AVG.	S.D.	AVG.	S.D.
Increase	30.33	18.25	—	—
Decrease	10.00	2.89	15.20	9.66

Avg. = Average.
S.D. = Standard deviation.

readings following the application of pressure. In a general way these responses seem to follow Ohm's Law.

Table II shows the range of readings of the fifty-three cases studied. Where the amperage rose there was an average increase of 30.33 microamperes with a standard deviation of 18.25 microamperes. In the cases where the amperage dropped there was an average decline of 10.00 microamperes with a standard deviation of 2.89 microamperes. When there was a drop in voltage the average decline was 15.20 units of voltage with a standard deviation of 9.66.

While the combination of amperage and voltage readings was altered after pressure was applied to the teeth, it showed a tendency toward an increased irritability of the pulp as denoted by the lowered threshold of stimulation.

In summarizing some of the main points of this report the following are of significance:

1. Histologic investigators are not in agreement as to the changes which occur within the dental pulp, be they of a temporary or permanent character, after teeth have been subjected to mechanical pressure.

2. Many of the investigators conceded some pulpal change ranging from simple hyperemia to decided degenerative changes.

3. From a clinical standpoint it is conceded by many that traumatized teeth may not reveal immediate recognizable clinical or radiographic signs indicative of pulpal change. In fact, these signs may not become apparent for a long time following the initial injury. It has been expressed by some men that where pulp disturbances have followed in the wake of orthodontic treatment, a previous injury to the tooth was responsible for the condition.

4. Many men are in agreement with the fact that severe pressure will cause pulp devitalization.

5. By the use of the electric current in appraising pulpal changes as the result of pressure, it was found that the threshold of stimulation was lowered, which is indicative of pulpal irritation.

In conclusion, it may be stated from the foregoing that proper and accurate pulp-testing, especially of the upper four incisor teeth, should be of value to the orthodontist to determine the status of the pulps prior to treatment, thus safeguarding the patient and the orthodontist.

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CENTRAL MEDICAL BUILDING.

Round-Table Discussion

Cephalometrics

Interest was manifested in cephalometrics and what it contributed to a correct diagnosis. My answer to this was that it was like all other aids to diagnosis, not perfect or sufficient unto itself but the best single diagnostic aid known at the present time.

My subject dealt with the relation of the teeth to the basal bone as an indication for or against extraction. I pointed out the following:

1. Faces, therefore the maxilla and mandible, seemed to be getting smaller down through the ages while as yet the size of the teeth has not shown much change.

2. The only unquestioned reason for extraction was a condition where the teeth were too large for present or predicted jaw size.

3. A second reason for extraction was a condition in which the teeth had migrated or drifted mesially too far with respect to the basal bone, and the orthodontic mechanisms could not be operated to place them adequately in the desired positions.

4. Only by seeing the bones and teeth as revealed by the cephalometric roentgenogram could it be determined where the teeth were in relation to the basal bone and how their size compared with the amount of basal bone.

5. Casts (which could not include basal bone), even though sectioned, could not show *position* of teeth with respect to the basal bone even though they might indicate their *inclination*. The angle formed by the intersection of the Frankfort and mandibular base planes did not indicate the *length* of the body of the mandible nor the *position* of the teeth superimposed upon it.

6. Extraction was indicated if the teeth were too large for their basal bones or could not be placed in correct relation to the basal bone because of certain orthodontic limitations. (This is a conclusion drawn from *1, 2,* and *3.*)

One question asked was this: Is it not true that the reason some orthodontists do not extract premolars is that they fear a dished-in or edentulous facial profile as a result of treatment? Answer: Undoubtedly, and some get just such a result because they have heard so much said about moving the incisors back onto the ridge that they move them too far lingually instead of moving them to their correct position and then completing the space closure by carrying posterior teeth mesially. One other reason for this pronounced lingual movement of the lower incisors is the statement that, in all cases of Class II, Division 1 (Angle), the lower incisors are too far forward on their basal bone.

The Round-Table Discussion was held at the American Association of Orthodontists Meeting, Broadmoor Hotel, Colorado Springs, Colorado, Sept. 30–Oct. 3, 1946.

This is not true; in fact, very few can be shown to be positioned too far forward although their axial inclination might lead one to assume this.

It was then pointed out that the profile cephalometric roentgenogram shows the labiolingual thickness of the mandibular basal bone very clearly and in the roentgenogram you can see the labial and lingual compact outer layer and the internal cancellous bone just as though the mandible were sectioned in the midsagittal plane. Thus, the relation of the incisors to the cross section of the mandibular basal bone can be seen.

L. B. HIGLEY, Chairman.

Photography in Orthodontics

The following orthodontists took part in the round-table discussion "Photography in Orthodontics":

N. G. Gaston, Monroe, Louisiana
C. Roy Brooks, Detroit, Michigan
Ernest T. Klein, Denver, Colorado
L. M. Waugh, New York, New York
David L. England, Santa Barbara, California
Everett W. Bedell, St. Louis, Missouri
Robert E. Bedell, St. Louis, Missouri
George H. Herbert, St. Louis, Missouri
W. C. McCarthy, Anaheim, California
Cecil W. Neff, San Diego, California

The subject was introduced when chairman Will G. Sheffer, San Jose, California, passed around a registration questionnaire which, when completed, furnished information about the type of cameras used in the office or for pleasure, and whether developing was done commercially or by the orthodontists themselves.

With this information, the discussion then led to types of lenses, advantages of fixed-focus cameras over the use of the ground glass; fine-grain time and temperature vs. drugstore type development; and the advantages of exacting photostatic technique over diffusion during enlarging.

Such subjects as stereoscopic photography, portraiture lighting, intraoral photography, darkroom technique, and finishing of prints were discussed. Each man described his office photographic setup and received helpful suggestions as the many interesting questions were asked.

The group was going strong, delving into photographic formulas, when time was called. The discussion could have gone on most of the afternoon.

WILL G. SHEFFER.

REPORT OF THE COMMITTEE ON PRESIDENT'S ADDRESS

The Committee on President's Address has read with studied interest the splendid presentation offered at the opening session of this meeting.

We heartily concur in the words of praise for the members of the Army and Navy Dental Corps for their outstanding service during the recent conflict.

A note of concern was expressed in regard to the National Health Bill and we are certain that each member of our association is in agreement with the thoughts expressed on this subject.

We are also heartily in favor of a closer relationship with our Latin-American confreres, and join in the expressed hope that another International Orthodontic Congress may be arranged.

The committee considered the proposed changes in the policy and methods now in effect in the matter of the Ketcham Award. It is our opinion that the magnitude and significance of the award is such that, as in the past, the greatest of care should be exercised in the selection of each recipient. We also strongly feel that unless a candidate of outstanding scientific attainment is available at the time of the annual selection, the lofty ideals of the awards should not be compromised and no awards should be conferred in that year. The suggestion made that it be an award to encourage young men to higher scientific attainment seems to be in conflict with the annual award of the Research Committee which has been developed for just such a purpose. The suggestion relating to the committee for the selection of candidates for the Ketcham Award seems to us to be a matter which should be considered jointly by the Directors of the American Board of Orthodontics and Board of Directors of the American Association of Orthodontists.

The committee concurs in the President's recommendations concerning a nominating committee for the election of officers. This matter has already been discussed by the Board of Directors and a report has been formulated by the Committee on Constitutional and Administrative By-Laws, and this report has been presented at this meeting.

Respectfully submitted,

STEPHEN C. HOPKINS, Chairman.
HOMER B. ROBISON
EARL G. JONES

Presented at the meeting of the American Association of Orthodontists, Sept. 30-Oct. 3, 1946.

REPORT OF THE NECROLOGY COMMITTEE

MR. PRESIDENT AND MEMBERS:

Our Eternal Father, who sees fit to create and take unto His own, has claimed from among our members the following friends, cherished by us and missed by their loved ones:

Melvin D. Baird	Spokane, Washington
Floyd E. Gibbin	Buffalo, New York
A. C. Gifford	Oshkosh, Wisconsin
Herbert C. Hopkins	Washington, D. C.
Carrol C. Johnson	Memphis, Tennessee
Harry E. Kelsey	Baltimore, Maryland
Frederick E. Leslie	Oakland, California
Eda B. Schlencker	Rochester, New York
Emilo Velutini	Philadelphia, Pennsylvania
Virgil D. Westcott	West Vancouver, B. C.
Seymore L. Winslow	Santa Rosa, California
L. H. Wirt	South Bend, Indiana
F. L. Stanton (retired member)	New York, New York
Ira B. Stilson (retired member)	Providence, Rhode Island

Through their early lives the determinate factors through which each of them saw fit to embrace the profession of dentistry was a heritage which belonged to them—each in his own right. That they should have chosen orthodontics as the specialty for their careers has long been justified by the noble records they left behind. Their lives were such that in their passing we can all revere them and hold them as patterns to follow.

It would be the desire and intention of your Necrology Committee to have prepared individual resolutions for our departed members. However, the interval of time between our last meeting in 1944 and the present session, caused by reasons beyond our control, makes it advisable to submit herewith the individual records of our late members and to offer the following resolution:

WHEREAS, The American Association of Orthodontists in its Forty-Third Official Session at Colorado Springs, Colorado, on Oct. 2, 1946, places itself on record as missing these dear departed members and pauses in silence for one minute in memory of their contributions to orthodontics and to this Association.

BE IT RESOLVED, that the American Association of Orthodontists recognize in this brief tribute its debt to, and appreciation of, their work.

BE IT FURTHER RESOLVED, that this record be added to the minutes of the Association and copies be sent to the families of our departed members with our heartfelt sympathy.

LEIGH C. FAIRBANK, Chairman
A. B. THOMPSON
W. T. CHAPMAN

Presented at the meeting of the American Association of Orthodontists, Sept. 30-Oct. 3, 1946.

REPORT OF THE EDITORIAL AND PUBLICATION BOARD

To the Board of Directors, American Association of Orthodontists:

It becomes my duty in the absence of Dr. E. C. Lunsford and as the senior member present, to report for the Editorial and Publication Board.

It has been found upon investigation that never have the conduct and affairs of the Orthodontic Section of the American Journal of Orthodontics and Oral Surgery been conducted on such a high and satisfactory plane. In this connection, the relationship between the American Association of Orthodontists and the publisher has been most cooperative and cordial. It has been found that, in matters of general management, editorial policy, and control of advertisements, they are entirely within the hands of the Editorial Board, the publishers themselves having nothing whatever to do with it.

Upon further investigation it has been found that the five-year-old agreement entered upon in 1941 between the American Association of Orthodontists and The C. V. Mosby Company should be changed sufficiently only to bring the contract itself up to date, and in accordance with the present-day methods and practices, which have been found to be the most satisfactory to all.

In making the changes to bring the old contract up to date, the original clause for cancelling the contract by either party upon one year's written notice remains the same. Sectional Societies, instead of being listed, have been referred to only as component or constituent societies. This was done in case any of these societies should decide to combine or to change their name. It has also been suggested that the editors elected to represent the different Sectional Societies should be known as "Sectional Editors," and that these elected representatives and the Editor-in Chief would constitute the Editorial Board and assume full responsibility for the Journal. These men hereafter to be known as Sectional Editors could then appoint such Associate Editors to fill out any staff requirements such as the Department of Abstracts and Reviews, Consultant in Children's Dentistry, Pediatrics, etc.

The new contract provides that when a Component Society elects its own Editor, whether annually or for an indefinite term, he is then installed as their personal and direct representative and does not have to be approved either by the American Association of Orthodontists or The C. V. Mosby Company. This is exactly the plan as it has been in operation during the last five years, and for the life of the present agreement, which (i.e., the agreement) has not been carried out as specified, but has been found to be satisfactory. It has also been decided to eliminate the provision that The Mosby Company would in any way be consulted or in any way have a right to disapprove the appointment of any new Editor-in-Chief. It is believed that with the right to cancel the contract with one year's notice and the fact that there have been thirty-two years of most cordial relations between the American Association of Orthodontists and The Mosby Company that any such clause as this is unnecessary. It is also felt that this pertains in the same way to the matter of any financial arrangements between the Editor-in-Chief and The Mosby Company for secretarial assistance, postage, etc.

This new contract has the unanimous approval of John C. Mosby, Chairman of the Board of The C. V. Mosby Company, Dr. Claude R. Wood, Vice-President of the company, and Dr. H. C. Pollock, our Editor-in-Chief. Again may it be stated that this change has been brought about mainly to bring the contract itself up to date and in accordance with existing methods by which the Journal is being conducted, and which, under the new contract, are not and will not have to be changed. Let us hope that this contract will be unanimously accepted by the Board of Directors of the American Association of Orthodontists.

Respectfully submitted,

Stephen C. Hopkins, Acting Chairman

Presented at the meeting of the American Association of Orthodontists, Sept. 30-Oct. 3, 1946.

Editorial

An Anthropologist's Message to Orthodontists

IF YOU have read Weston A. Price's book, *Nutrition and Physical Degeneration,* which was written in order to record his clinical observations made on primitive people in various parts of the world, you noted that he believes that no problem is so important to modern civilization as the finding of the reason for the obvious narrowing of the face and dental arches that afflicts a considerable proportion of modernized people, contrasted with the arches and facial forms of primitive peoples.

In reading Price, you soon sense that, as a result of his wide clinical experience, he believes that much of this is due to prenatal as well as to postnatal nutritional conditions. In a word, it is largely a matter of food balance of the pregnant mother and also of the offspring subsequent to birth. You will note that he also believes there is a tie-in between poorly developed and small pelvic bones and the same condition as manifest in the bones of the face and jaws.

To the everyday practicing orthodontist the approach to these problems of men in collateral fields never ceases to be a source of great interest. Orthodontic program committees are forever searching for scientific thinkers who can give a little more light on the subject.

Commenting that "realization of ignorance is the beginning of wisdom," Prof. Earnest A. Hooton, of the department of Anthropology of Harvard University, proved to be a very interesting essayist at the Colorado Springs meeting of the American Association of Orthodontists, Sept. 30, Oct. 1, 2, and 3, 1946, in that he stresses many other important angles to the subject.

He brought out that man is the creator of numerous gadgets of all kinds and that he now is in grave danger from some of the gadgets that he has created; and there can be no doubt about it. He observed that man, for the most part, has tried to substitute the use of machines and gadgets for the proper exercising and performance of work by his own body. Man uses powerful machinery for the grinding of his food, chemistry produces soft foods that need no grinding by the teeth, and then man wonders why his face, which is the stage setting for his teeth and jaws, get smaller as the generations go by. Such was the gist of some of his remarks, bringing out that functional stresses are important to dental and facial form and size.

Dr. Hooton then followed with the observation that orthodontics is an applied science which has evolved largely as a result of the necessity of correcting so-called "crooked teeth," and that it has made a pretty good record in spite of its limitations. In doing this, the orthodontist has found, however, that not only the teeth needed correction, but many times the whole body has something wrong with it, and that orthodontic treatment constitutes only one

of the needs for biologic rehabilitation of an entire body. So reasoned Dr. Hooton, and there can be no contention that such is not exactly what has happened in orthodontic evolution.

When they realized this, he went on, orthodontists turned to other sciences for help, for it did not take them long to understand that if they stuck to mechanical devices alone, the entire perspective and progress of the work would begin with the teeth and end there. The orthodontist turned to the geneticist and to the physiologist, wanting to know why malocclusion ties in as a symptom with hypothyroidism. He tried questioning various and sundry other sources, only to become confused. The endocrine work by Howard and others opened the orthodontist's eyes to a horizon far beyond the teeth and jaws, and made him realize that there is much to orthodontics beyond the oral cavity. This endocrine work called the attention of the medical profession to the fact that malocclusion is sometimes a classical symptom in a syndrome involving the entire skeletal structure.

The growth studies of the Brush foundation of Cleveland, to which Broadbent made important contributions pertaining to the growth of the face as related to the teeth, were mentioned as highly important to the over-all picture.

Incidentally, Dr. Hooton paid tribute to the work of Broadbent, Hellman, Margolis, Baker, and Brodie (all orthodontists), and he stated that they have contributed more to scientific knowledge of the growth of the face, teeth, and their interrelationship, than have anthropologists and other growth workers, and that the next step forward in solving some of the orthodontic problems is plain. It is the widespread use of the plan of study started by the above workers in collaboration with anthropologists, pediatricians, and anatomists, in order to study the problem of total growth as related to the anatomy of parts in which orthodontists are particularly interested.

The impression is quickly gained in listening to this authority on anthropology that it is his view that the problem of malocclusion is primarily both evolutionary and pathologic, and that in order to treat malocclusion intelligently much more knowledge needs to be spaded direct from the grass roots by comprehensive research of a high degree of efficiency.

Other impressions were gotten; for instance, it seems that the orthodontist must decide as soon as possible whether in attempting to correct malocclusion he is combating a natural evolutionary trend or whether he is attempting to remedy a pathologic condition that is the result of infection or atrophy of function arising from too much dependence upon soft cooked foods, of subnormal bone development due to nutritional defects, or of all of these factors in combination.

While paying respect to the mechanical dexterity of orthodontists, he leaves little doubt in the mind that, in his opinion, there is a limit to the possibilities of orthodontic treatment from a retention standpoint and that there is a wide etiological field as yet practically unexplored.

Such teachers and their views are good for orthodontists because it gives the viewpoint of a trained objective scientific mind from the outside looking in. It keeps the workers thinking.

When Dr. Hooton suggested that orthodontists must be the spark plug in starting a comprehensive research problem on a level far and above the possibilities of the private practitioner in order to solve their problems, he no doubt suggested the only practical way these problems will ever be solved during the present generation at least.

It can be added that it is amazing how far orthodontists have gone in the early years with what might be called, for the sake of a better descriptive term, a "popular mechanics" perspective of treatment and a very limited concept of etiological factors. After listening to Dr. Hooton, orthodontists will still be proud of their accomplishments, because they will more fully understand that they have gone so far with so little.

It requires a fine technician to do a good job of correcting malocclusion, but added to that he should have a better knowledge of a number of other component influences.

Hooton's lecture and its publication will add much to constructive orthodontic thinking, but just exactly what it is all about from a primarily scientific standpoint, Hooton admits that there is, up to this time at least, little but supposition and speculative thinking upon many phases.

H. C. P.

Health Insurance

COMPULSORY health insurance would harm rather than improve dental health inasmuch as it would place the nation's health service in the hands of those who are not trained in the health professions."

This was the gist of remarks recently made by Dr. Sterling V. Mead of Washington, D. C., president of the American Dental Association. He put the American Dental Association on record as being opposed to any health plan where a portion of the earnings of the people would be used for inferior service and for the benefit primarily of political-minded bosses.

This objection is not new, but its repetition again by the president of the American Dental Association is a sharp reminder that there is danger of the passage of the Wagner-Murray-Dingell bill in the Eightieth Congress, and that there are strong forces at work to pass such a bill.

This bill proposes socialized medical care at a cost of a probable 3 to 6 per cent deduction on the wage earners' pay. That, added to the present social security law on salaries and wages, will be a sizable amount.

It seems, at this crucial period, that ways and means to offset this bill have not been at all effective. There is little doubt that the nation needs better medical and dental services for all of the people; however, it is ineffective to stop this movement simply to cry, "It's Socialism," and make no further practical moves to end the increasingly manifest momentum.

The American Medical Association took an inadequate step forward in announcing a voluntary medical care plan organized on a regional basis. If the Wagner-Murray-Dingell bill is not to be passed in some form or other, substitutes heretofore not on the horizon will surely have to be created, and time is growing short.

Correspondence

OPEN LETTER TO ORTHODONTISTS

Dental practice, including orthodontics, is an integral part of present-day health service to the nation. Hence in recent years, not only have various activities of research been directed in the search for the cause of dental disease and factors of use in its therapy, but the farsighted leaders of the profession have been concerned with how many dentists we have, at what rate they are being graduated, and at what rate they are retiring from professional activity. Accordingly, we have noted studies on dental man power that is now available and that may be made available in the future.

Interest has been directed also toward the magnitude of the problem of rendering adequate dental service to our population. How many fillings, extractions, crowns, bridges, dentures, and how much orthodontics do the people need, how much do they purchase, and how many dentists, laboratory technicians, chair assistants, and how much equipment would we require if everyone who needed care was in a position to request it?

The orthodontist himself should be in a position to provide adequate data relative to his part of the general picture. How many cases of malocclusion exist among our 33 million children? What types of malocclusion can be expected per 1,000 children encountered? And among those with undisputed malocclusion, how much orthodontist-time is required per case of given type and what varieties of appliances are used per given type of case?

There is little disagreement with the view that we possess very little, hardly any, quantitative data and statistics on these questions. We, as orthodontists, should be the first to make them a reality. This, of course, cannot be done all at once. But the direction of our thoughts toward these objectives should be accompanied by activity and research. The collection of such information will accelerate the availability of these much-needed data.

The collection of such material can be accomplished, on the one hand, by careful record keeping in fact-finding clinics established specifically for that purpose, and, on the other hand, by questionnaire survey of orthodontists now in practice. The fact-finding clinic now operating at Sturgis, Michigan, through the cooperation of the Department of Orthodontics, University of Michigan, and the Dental Division, Michigan State Department of Health, represents only a beginning in the approach to fact-finding through examination of random samples of children.

The questionnaire survey method also can give valuable information which, when assembled, provides insight into the problem not obtainable by observation in any single orthodontist's office.

I would like to suggest, therefore, that the questionnaire survey method be applied, and that orthodontists in all parts of the country unanimously support such an activity. General dentistry has done it, pediatrics is now doing it on a large scale, and orthodontics is a sufficiently important arm of health service to warrant such an activity within its ranks.

Specifically, we at Michigan are collecting information in a fact-finding clinic with regard to the kinds of cases occurring and treatment procedures required for a random sample of school children. We would like to round out our research by collecting data on types of cases encountered and kinds of treatment available over the country as a whole. We would like to propose for this

purpose a questionnaire survey of orthodontic practitioners. It is our hope to be able to design a questionnaire which is brief and to the point, send it to you, and have you answer the questions and return the form to us here. *Can we count on your cooperation?*

Please answer "yes" or "no," or write your opinion on this question to Dr. George R. Moore, Head of Orthodontic Department, School of Dentistry, University of Michigan, Ann Arbor, Michigan.

GREETINGS FROM SAN MARCOS UNIVERSITY, LIMA, PERU*

The kind invitation to write a few words with which the staff of the University of Michigan Department of Orthodontics has favored me affords me the happy opportunity and honor of writing to an institution which I have come to regard as one of the most outstanding universities of the United States, an honor which I accept and for which I thank this staff on behalf of Peruvian dentistry.

In my opinion, international scientific visits have a double purpose: to create professional bonds among colleagues in different countries who are striving for the advancement of their respective sciences—in our case, dentistry—and to stimulate the attainment of perfection in their several scientific and social aspects.

We should take pride in saying now that Pan Americanism, for which so many eminent men have labored—among whom we should remember the great President Roosevelt with veneration—is a reality today; we must persevere by collaborating, each within his own plane of professional activities and aptitudes, incorporating into the daily plan of our life and work the perfecting and the strengthening of this Pan Americanism.

My visit to the United States on invitation of the Department of State is due especially to my interest in the program of dental aid for children, both in general dentistry and in orthodontics; for, as you well know, the practice of orthodontics is limited to the people who can pay for it, while very few receive free care. For this reason I immediately made contact with the universities in order to observe and study the teaching of orthodontics, and with public health institutes and philanthropic social aid establishments in order to become acquainted with their working methods and their organization.

The professor of orthodontics in the Faculty of Dentistry at Lima is Dr. Augusto A. Taiman. I propose to collaborate with him on my return in awakening a greater interest in this specialty among the members of the profession.

My teaching association with the University of San Marcos is in the department of Clinical Surgery, under Professor Fortunato Quesada. I am entrusted with teaching fourth-year medical students those things which are related to clinical surgery and dentistry. You are perfectly aware that orthodontics is a powerful aid in the techniques of treating fractures of the maxilla, not only in the placement of arch wires and ligatures, but also, still more important,

*This is a message from one who took advantage of the invitation of the Department of State of the United States to collaborate with the medical and dental professions in North America.

It will be recalled that a number of men among our neighbors to the south attended the meeting of the American Association of Orthodontists in New Orleans, and subsequently took advantage of postgraduate courses in some of our dental schools.

EDITOR

in the examination and classification of the articulation of the teeth; this last is unknown to physicians and, we may say without exaggeration, also to many dentists in general practice. This is the reason for my interest in studying these problems.

Dentistry, offshoot of medicine, now constitutes one of the branches of the medical science; it has won for itself the honorable position it now occupies through its powerful vitality, by dealing with problems for whose solution medical science itself, with its lofty criteria, has enlisted its aid; thus dentistry has become an important and frequently indispensable auxiliary in the prevention and treatment of disabilities of the whole dentofacial region.

The social function of the dentist no longer needs to be emphasized, because his role is as broad as the work he carries out is decisive and indispensable. The preventive and curative work of dental science precedes or accompanies that of the physician, and follows it up throughout the human life cycle—by attention to the mother during gestation; to the child during the school years; and, in short, throughout the unfolding of the biologic process.

With the passing of the period of speculation and philosophy in the great advance of medical science, dentistry has assumed an outstanding position in the last fifteen years. Today we are proud to say that it takes a principal role in scientific discussions and in pathologic diagnosis in general.

An eminent teacher has said, "The present period of clinical dentistry will occupy an outstanding place in the history of the profession, for two principal reasons: the constant and increasing interest we dentists have in diagnosis and, second, the effort of the more progressive groups to elevate and broaden the field of clinical dentistry." In accordance with this clear statement, the dentist of the present and of the future should assume the role of specialized surgeon of the buccal cavity. We remember the interpretive words of Roberts, who said, "Dentists do not know enough medicine and medical men do not know enough dentistry."

Therefore, I maintain that a broad medical education should be the basis of teaching in all progressive dental centers.

The dentist is and should always be not only a clinician but also an artist. The fundamental aim of modern dentistry should be to maintain this balance.

I cannot do less than to mention with special praise the Department of Orthodontics of the School of Dentistry of the University of Michigan which is known for its accomplishments beyond the borders of your country. Among all of the universities I have visited, this is one of those to be admired.

In conclusion, let me thank the Latin American and the North American dentists who together have attended this meeting, as I express my great hopes for fraternity and mutual greatness in the Americas.

JOSÉ SANTOS HERRERA.

Department of Orthodontic Abstracts and Reviews

Edited by

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Maxillo Facial Injuries in Children and Their Sequelae: By G. J. Parfitt, M.R.C.S., L.R.C.P., L.D.S., *D. Record* **64**: 159-168, July, 1946.

Maxillofacial injuries in children have considerable interest for the orthodontist. There are two interesting features in the study of injuries in the child; the difference in healing as compared with that of an adult, and the effect of such injury on growth fulfillment.

Forty or fifty accident and air-raid casualties and some few osteomyelitis cases were treated alongside similar adult cases. I would say that the child heals at least twice as quickly as the adult. This belief enjoys universal acceptance. There are two end results to bone healing, union and nonunion, and tissue breakdown and sequestration occur as rapidly as does uneventful healing; in fact, the child's tissues heal just as rapidly wrongly as rightly. On the one hand, a compound fracture heals with remarkable speed and tissue survival, with but little local reaction; on the other hand, a relatively mild injury breaks down with quantities of pus formation, leading to early sequestration as well as early absorptive bone loss, a condition of the state of healing termed nonunion.

One case of a girl of 12 years showed x-ray appearance in the fracture area of nonunion thirty-five days after the injury. This appearance consists of rounding of the bone ends with the characteristic space between the rounded fragments. Such an appearance and diagnosis in an adult would be out of the question before perhaps six months after injury and, in this particular case, at the time were thought to be most unlikely, so that the fragments were immobilized for a further four months. The apparatus used was a cap splint to the teeth on each fragment connected by a bar which bridged the fracture, so allowing freedom of movement of the mandible without intermaxillary fixation, but rigid immobilization of the two fragments. At the end of this four-month period, the original diagnosis was confirmed, and after a further waiting period dictated by conditions other than the fractured jaw, a graft had to be done. The graft took uneventfully and with excellent speed.

In addition to the difference in the speed of reaction of the child's tissues there is a high tissue resistance to infection, but when infection does take place there is a severe reaction to it. It was felt also, from the examination of wounds, that the young bone must be more resistant to fracture. Injuries were seen in the soft tissues which laid bare the mandible over considerable areas, and this protruding into the dressings was found to be unfractured, and moreover after gentle cleansing of the wound and suturing the soft tissues back into place, the bone survived uneventfully. No doubt these points were forced into relief by the great minority of injured children treated, and had the numbers been reversed, no doubt the wearisome undramatic healing of the adult fracture which we consider miraculously fast, would have been remarked upon.

Teeth involved in the fractured bone can be frequently retained in the child, but should infection occur about them, more damage results. Some teeth clinically involved in fracture were unaffected; they continued to erupt into place and form roots correctly. Others involved continued to erupt but did not form roots.

Some teeth involved in fracture become apparently arrested in growth. The surrounding bone and teeth continue growing and erupt, leaving the injured teeth increasingly behind in the jaw at a considerably lower level. One such tooth was finally exfoliated practically four years after injury.

A bone grows and increases in size both by bone formation upon its ends and in thickness by deposition by layer upon layer to its sides. In the main the growth of the mandible can be so described. It is of fundamental importance to understand that there is no interstitial growth in bone; no increase in length occurs by growth in the middle, but by addition to the ends only. Not that the central area of a bone is inert, for it is actively changing its pattern and structure, but such activity does not contribute to the increase in length.

Only two things can happen to any given element of bone after it has been laid down, either more layers of bone are laid upon its surface or it is absorbed at its surface.

A purely local injury to the middle of a bone may interfere with the local increase in thickness, whereas an injury to the growing end may interfere with its increase in length, producing considerable deformity. Both these sequelae are found in the mandible following upon injury. Growth changes of the mandible are more complex than those of a long bone, both because of its shape and because of the presence of teeth. Local growth interference following injury may show as local lack of increase in thickness and also distributed tooth formation and eruption.

A child of 9 years was bitten by a dog and fell, sustaining a fracture in the first molar region. This was neither immobilized nor drained for twenty-one days. The fragments were then immobilized, the abscess drained, and the necrotic tissue and loose teeth removed. The operation was followed by prolonged immobilization, sequestration, and nonunion, and then a period of rest with all appliances removed. This rest was at that time our custom before bone graft operation. During this rest period the posterior fragment swung far forward. By superimposing the tracings of serial x-rays taken during immobilization upon the shadows of the splints, the premolars were shown to be erupting, and the lower border apparently showed little change, and finally, upon obtaining the x-ray after the splints had been removed for some months and superimposing this upon the tracings of the previous lower borders, the posterior fragment was shown to have swung far forward. There was some justification in using the splints as a fixed point for the superimposition of x-rays, for these were cemented to the lower incisors and also fixed to the splints cemented to the upper teeth.

After this rest period had elapsed, the bone graft operation was performed. A block bone graft from the hip was inserted in the ununited fracture area and was held in place by two stainless-steel wires passing through the graft and fragments. These wires were left permanently in situ.

These two wires form, possibly, a unique superimposition point for fracture x-ray pictures in a growing child, and repeat the classical experiments of Duhamel and John Hunter, in which lead shot and wires were inserted into the limbs of animals and birds in order to study their growth. The value of these wires for superimposition of x-rays lies in the fact that as there is no interstitial growth they will not move apart on that account. They were 1 inch apart. The only way that these wires can move is by involvement in fibrous

tissue contraction or by local abscess formation. Tissue changes external to the bone will have but little effect, for the wires were twisted tightly against the bone surface beneath the periosteum.

In the study of growth change, superimposition of x-rays upon points external to the bone may indicate growth changes as a whole but cannot show absorption or deposition of bone to any given surface; for such an estimate by this method, fixed points must be obtained within the bone itself.

The follow-up of this case now held great promise, except for the difficulty of obtaining subsequent x-rays with plate, head, and tube in identical positions over a period of months and years. This ideal is well-nigh impossible. The pictures I managed to obtain of this case vary; this does not make superimpositions of tracings valueless, but it does prevent any accurate measurements. The inaccuracy increases with the distance from the wires, the greatest accuracy being in the area between the wires themselves; in this case this is a featureless area. An assessment of the accuracy of the positioning of the x-rays can be made by noting the widely separated bony points on the plate, such as the right and left angles of the mandible and the hyoid arch, seeing that these make comparable patterns on the plates, and discarding those pictures which have not been taken sufficiently near to the identical angle as they are not comparable. Anteroposterior views are also compared, for, in the positioning of the patient for this shot, the wires and the condyle head are on a plane parallel to the plate and at right angles to the x-rays, a condition which minimizes inaccuracies due to positioning; in these condyle growth can be observed by again superimposing subsequent x-rays upon the wires.

Kelsey Fry pointed out years ago that on clinical observation the main growth of the mandible was the condyle area. This was from the study of cases of facial deformity where one condyle had been damaged and ceased growth, so producing a short ascending ramus. Martin Rushton has lately again pointed this out by a series of histologic studies of growing condyles. This case has brought this point well home to me.

Various points can be noted: The premolars have erupted and have changed their position in a curve, the center of which is situated posterior to the teeth. Increase of ascending ramus has been brought about by growth of the condyle. The angle of the mandible and posterior border of the ascending ramus have grown by surface deposition and the anterior border of the ascending ramus has been absorbed so that the front edge has moved distally. The third molar has changed its position but little during this time period and little change has occurred at the lower border, but the relative positions of the third molar and the anterior border of the ascending ramus have altered. In the early tracing the ascending ramus if projected would pass in front of the mesial border of the tooth, whereas in the later tracing the projection of the ascending ramus would pass near to the posterior border of the tooth. The third molar has moved forward a little perhaps as the first and second molars have been extracted, but some of this change, I believe, has been brought about by absorption of the anterior border of the ascending ramus in this particular case. Molding changes of the graft area will also be noted. Another important detail is that the anterior wire itself has altered position during the first months, taking up a more vertical position and thereafter showing but little change. I believe this to have been brought about by involvement of fibrous tissue contraction about its upper end. It will be seen that the nonunion area of the alveolus mesial to the third molar has been left ungrafted and that the wire approaches this area of scar tissue; one year and three months later, this area had filled with bone and the wire had changed position slightly, swinging about its twisted ends situated at the lower border. The clinical assessment of this case is that the main growth area (the condyle region), remained

unaffected, producing little or no facial deformity in spite of the severe local injury, over an extended period of time.

In a low-power microphotograph of a section through the head of the condyle of a child, the growing cartilaginous cap can be seen with bony replacement beneath it, which provided the growth addition to the bone. Absorption can be noted anteriorly in the neck of the condyle at the site of the muscle attachment, perpetuating through the growth changes the shape of the condyle head and neck.

My next case is one of osteomyelitis of the mandible involving the condyle. A child of 6 years was admitted with osteomyelitis following upon abscess and tooth extraction, and, after three operations for drainage, sequestrated a large portion of bone including the angle, the ascending ramus, the distal two-thirds of the condyle itself and the second molar.

In the lateral x-ray of the affected side three months after tooth extraction, the distal two-thirds of the condyle could be seen to have separated from the anterior portion. After a further two-month drainage period, an excellent local recovery ensued, the whole area being replaced by bone, but I am afraid the remaining condylar tissue will be unable to keep pace with the opposite side and I expect therefore some deformity will result as the child grows up.

Deformity is produced by such disease and discontinuance of growth of one condyle. I have examples of ankylosis of one or both condyles following osteomyelitis, after fall, after scarlet fever, after mastoiditis, and after condylectomy, and in one case the original disease was unknown. The deformity in unilateral disease is a short ascending ramus upon the injured side with displacement of the chin to that side, and the consequent flattening of the face upon the opposite side. This causes gross asymmetry. With bilateral disease there is symmetrical bilateral underdevelopment of the lower jaw.

A case of osteomyelitis following abscess upon a tooth at the age of 4 years shows a compensatory growth effort of the angle region of the mandible in a downward direction. This area is the muscle attachment and this growth effort has been noted to a lesser degree in several cases. Photographs and x-rays were taken nineteen years after the disease process, the patient being then 23 years of age.

This series of individuals was examined ten to twenty-five years after the original mishap and showed to some extent this deformity. I have heard of a case of gross asymmetry such as this following simple fracture of the condyle without infection; and so with some misgiving I again followed up some of our condyle fractures in children, for if the condyle is so misplaced, growth must surely suffer. Fortunately, however, no such serious case had come our way and the examples of condyle fracture have continued without deformity. The injury in these cases cannot have interfered with the growth in this region.

The Lamina Dura in Roentgenographic Interpretation: Changes During Tooth Movement; By Maury Massler, D.D.S., M.S., Chicago, Illinois, *Angle Orthodontist* **15**: 3-17, January and April, 1945.

The lamina dura is a term commonly used by the dental roentgenologist to describe that narrow portion of the alveolar bone which borders upon the periodontal membrane and which appears as a well-defined radiopaque (white) layer in the roentgenogram.

The purpose of this paper is (a) to investigate the significance and the structural characteristics of the lamina dura, (b) to describe changes in its appearance during various types of tooth movements and in systemic diseases, and (c) to point out the value of such knowledge in roentgenographic interpretation.

About 250 full-mouth, and a larger number of single, roentgenograms were obtained from the files of the Child Research Clinic. Fifty roentgenograms were of patients in the Department of Orthodontia at the University of Illinois, College of Dentistry. The roentgenograms were of patients from 2 years of age to adulthood and were examined with an ordinary hand lens at a magnification of about six times.

A study was also made of the roentgenograms and histologic sections of sixteen human jaws ranging in age from birth to adulthood.

The relative density or radiopacity of the alveolar bone was judged by comparing it with the dentin in the same film. The thickness of enamel and dentin in a given tooth is fairly constant and does not vary greatly in different individuals (Schour and Massler, 1940). While the calcification of these structures during their formative period is extremely sensitive to metabolic upsets, once formed and calcified, the enamel and dentin, because they are avascular, cannot and do not lose their calcium. The density of the enamel and dentin in the roentgenogram has been shown to remain comparatively stable under even relatively severe systemic conditions (Albright, Aub, and Bauer, 1934). The bone, on the other hand, readily reflects disturbances in mineral and systemic disease, and, in the roentgenogram, varies greatly in absolute and relative density as well as in structural characteristics.

Since the relative density of the enamel, dentin, and pulp is fairly constant, these structures can serve somewhat in the nature of a densiometer in a given intraoral roentgenogram.

A statistical evaluation of the full-mouth roentgenograms of about 300 individuals from 2 to 50 years of age showed that the appearance, the structural characteristics, and the degree of the radiopacity of the lamina dura depend upon the *rate of the eruption* of the teeth and changes with each stage of eruption.

From the moment the bony crypt appears in the roentgenogram until the tooth is in full clinical occlusion, a thick and distinctly radiopaque border is present on the bone immediately surrounding the tooth. At the same time, the periodontal membrane tends to be rather wide. The radiopacity of the lamina dura around the actively erupting tooth is as great as the much thicker and more highly calcified dentin in the same film.

As the tooth comes into full clinical occlusion and the rate of eruption slows down, the lamina dura becomes thinner and much less radiopaque. The periodontal membrane also becomes thinner. This is particularly apparent when the teeth of the same individual are examined roentgenographically over a period of time. One can easily follow the gradual loss in the thickness and the radiopacity of the lamina dura in the radiograms as the clinical eruption of the tooth progresses. The lamina dura persists as a distinct entity but its radiopacity tends to approach that of the surrounding bone.

Thus it was observed that the lamina dura is thicker, more radiopaque, and associated with a wider periodontal membrane in actively erupting teeth. These characteristics diminish as a given tooth comes into clinical occlusion and the rate of tooth eruption slows down.

Changes With Age.—The thickness and radiopacity of the lamina dura continue to diminish after the tooth is in full clinical occlusion and as the individual becomes older, but normally does not disappear altogether. The lamina dura is present but not very prominent in the roentgenograms of healthy older individuals with good occlusion. This can be correlated with the fact that the eruption of teeth and the growth of the alveolar bone are continuous processes and normally proceed throughout the life of the individual, although the rate decreases constantly (Gottlieb and Orban, 1938).

Lamina Dura in "Elongated" Teeth.—The removal of an antagonistic tooth allows the "elongation" or supraeruption of a tooth. In these cases the lamina dura becomes markedly accentuated in thickness and radiopacity during the period of occlusal movement. This occurs even in elderly individuals whose roentgenograms normally show a very thin lamina dura.

The lamina dura is absent about teeth which are embedded or so ankylosed that they cannot migrate.

Changes in the Lamina Dura During Orthodontic Tooth Movement.—Teeth often tend to shift quite markedly as a result of broken contact points resulting from interproximal caries. For example, when caries has destroyed the distal side of the lower first permanent molar and has thus broken the continuity of the arch. As a result, the first molar is tipping distally about a fulcrum near the apical third of the root. The lamina dura is thick and prominent and the periodontal space is wider at the mesial surface of the root in its gingival two-thirds. This corresponds to the area of apposition of new bone in histologic section as has been shown by Kronfeld (1939). The distal surface of the root shows a decreased width of the periodontal space and the lamina dura is thin and almost absent. This corresponds to the area of resorption. On the other hand, the second molar is migrating mesially to occupy the space left by the broken-contact area on the carious first molar. The lamina dura is thick and prominent and the periodontal space is wider along the distal, and the lamina is absent along the mesial, root surface. These areas correspond histologically to the areas of new bone formation and bone resorption, respectively.

Orthodontic Tooth Movement.—An even more accurately controlled and truly experimental analysis of the relation of the lamina dura to tooth movement and the formation of new bone is offered during orthodontic treatment. Rehak (1935) has made a careful analysis of the "Roentgenographic Interpretation of Bone Changes in Orthodontic Tooth Movement." The following findings are in complete agreement with those published by Rehak.

Before orthodontic treatment is instituted, most roentgenograms show a lamina dura of the usual thickness and radiopacity. During treatment and after tooth movement has been instituted, the roentgenogram reveals a lamina dura that is distinctly wider and markedly radiopaque on the bony surface from which movement is occurring (area of tension). The periodontal membrane is also distinctly wider on that surface. Thus the surface upon which new bone formation occurs as a result of the orthodontic procedure shows a wider and more radiopaque lamina dura. The surface toward which the movement is progressing (area of pressure) is the area upon which resorption occurs. The periodontal space is markedly thinner and the lamina dura is usually entirely absent along that surface.

During the period of retention following active tooth movement, the formation of new bone occurs upon the *entire* bony surface surrounding the tooth, including that surface of the bony crypt which was being resorbed during active treatment. Roentgenograms taken during the period of retention show a thick, distinctly radiopaque lamina dura around the entire root, particularly on the surface that was being resorbed *during* the treatment and is *now*, during the period of retention, an active area of new bone formation. Thus the roentgenographic picture of the lamina dura tends to become reversed during the retention period.

In roentgenographic interpretation and analysis of the lamina dura, the time and stage of tooth movement are therefore important. A prominent lamina dura indicated the area of *new bone formation* rather than the direction of movement. The direction of movement is also indicated, but only *during* active movement.

HISTOPHYSIOLOGIC CORRELATIONS

As has already been indicated in the preceding section, the area occupied by the lamina dura in the roentgenogram corresponds in histologic sections to the area of new bone formation around the teeth that are moving as a result of eruption, mesial drifting or orthodontic treatment.

Radiopaque Borders In Other Bones.—While we can only speculate as to the reason why the newly calcified alveolar bone is more radiopaque than the older adjacent bone, this phenomenon is not peculiar alone to the bone surrounding the tooth. A similar phenomenon is observed in the growing inner table of the bones of the cranial vault, the growing margins of the cranial sutures, and the growing epiphyses of the long bones. These sites of active bone deposition appear distinctly radiopaque and have also been described as "lamina dura" (Bernstein, 1933). In fact, the radiopacity of the epiphyseal plate during the period of rapid bone formation prior to epiphyseal union is used as a diagnostic sign of rapid bone growth when the radiograms of long bones are interpreted. Newly calcified bone, therefore, is radiopaque in any part of the body which can be observed radiographically.

It should not be supposed that *all* radiopaque borders are sites of new bone formation. In the interpretation of all radiograms, the effects of superposition must always be considered. However, the radiopacity produced by superposition and that associated with new bone formation are different and, with little experience, can easily be differentiated. The radiopacity produced by superposition is of a grayish quality and quite in proportion to the thickness of the structure. The radiopacity associated with new bone formation is distinctly greater, producing a much whiter appearance, quite out of proportion to the thickness of the structure.

CLINICAL CORRELATIONS

A good intraoral roentgenogram is to the clinician what a good histologic section is to the experimenter. Roentgenology is but a step from clinical microscopy.

The fact that the thickness and degree of radiopacity of the lamina dura in the roentgenogram can be used as a diagnostic sign of tooth movement is of value to the orthodontist and the pedodontist. The appearance of the lamina dura should also be of interest to the prosthodontist since it will reveal to him the possibility of tooth movement both before and after the placement of an artificial appliance or even a single restoration.

The teeth move constantly under the forces of occlusion so that the alveolar bone proper is constantly resorbing and rebuilding itself. This is evidenced in the roentgenogram by the presence of a definite lamina dura in even older individuals. The absence of a lamina dura, on the other hand, is indicative of a failure in the growth of the alveolar bone proper. This fact is of particular significance to the periodontist in both his diagnosis and prognosis.

The assessment of the health of a given individual is too often a subjective procedure. The correlation of systemic health with the growth of the alveolar bone may serve to indicate to the dentist the physiologic status of a given individual just as the roentgenograms of growing epiphyses of long bones indicate to the pediatrician the systemic status of his patient.

The following conclusions were drawn:

1. The character of the lamina dura was found to be directly related to the stage and the rate of eruption of the tooth which it surrounds. The lamina dura is very thick and characteristically very radiopaque during the active

eruption of the tooth. It appears on those surfaces of the bony crypt which histologically show active, new bone formation. The lamina dura of the roentgenogram is therefore the alveolar bone proper in the histologic section.

The thickness and radiopacity of the lamina dura diminish rapidly after the tooth is in occlusion and active eruption ceases. The rate of new bone formation also diminishes. However, the lamina dura remains continuous and visible, but narrow and only moderately radiopaque, as long as the tooth is in function. This is due to the fact that a moderate amount of new bone formation occurs throughout life in the alveolar bone proper. If the alveolar bone proper shows no activity, the lamina dura disappears. If the alveolar bone proper shows excessive activity (as in tooth elongation and tooth movement) the lamina dura becomes thicker and more radiopaque.

2. During orthodontic tooth movement, the lamina dura becomes much thicker and characteristically radiopaque on the side of tension and new bone formation. Histologically the newly formed bone shows a distinctly fibrous character. After the completion of the orthodontic tooth movement, the accentuated lamina dura becomes less prominent as the fibrous bone is reorganized into lamellar bone.

The side of pressure shows a disappearance of the lamina dura during orthodontic tooth movement. This is caused by the resorption of the alveolar bone proper. After the completion of the tooth movement (during retention) a *new* lamina dura appears on this surface as a result of the formation of new bone to re-embed the new periodontal fibers.

Roentgenograms and correlative histologic sections are presented to illustrate the various changes in the character of the lamina dura during various types of tooth movement.

3. After tooth movement of any kind, new bone formation occurs on the tension side of the bony alveolus. This bone is usually fibrous in character and makes the alveolar bone proper wider and thicker. Roentgenographically, this is seen as a thicker and more radiopaque lamina dura. The reason for the radiopacity is not known. After the fibrous bone is reorganized into lamellar bone, the lamina dura becomes thinner and less radiopaque.

4. Discontinuity in the lamina dura represents areas where the periodontal fibers are not embedded, and is caused by the failure in new bone formation or by bone resorptions as a result of local and/or systemic factors. A few examples are discussed.

5. The thickness and degree of radiopacity of the lamina dura in the roentgenogram can be used as a diagnostic aid to indicate the direction and amount of tooth movement and the character of the newly formed bone.

The roentgenogram is the clinician's microscope. The correlation between the appearance of the lamina dura and tooth movement should be of particular value to the orthodontist and the pedodontist. Its appearance in the roentgenogram can also be used as a measure of the health and disease of the periodontal tissues.

Age for Application of Orthodontic Appliances: Queries and Minor Notes, J. A. M. A. **130**: 825 (March 23), 1946.

To the Editor: What is the earliest age at which orthodontic work should be begun on a child?—M.D.

Answer: The earliest age at which orthodontic "work" should be undertaken is that time when there exists a condition which stands in the way of normal growth, development, or function of the teeth and dental structures and which has little or no possibility of being corrected by further growth or

developmental processes or by the institution of procedures which are simpler and less disturbing to the child.

It is seldom practical to place orthodontic appliances before the deciduous dentition is complete, although occasionally this has been done with satisfactory results. The nature of the particular problem must be the determining factor.

The correction of the condition may not prevent the occurrence of malocclusion from other causes at a later age and, in those conditions in which the malocclusion is due to discrepancies in harmonious relationships which arise from either genetic or growth and developmental causes, more than one period of orthodontic treatment may be advisable. Treatment in the early years in these instances is for the purpose of obtaining maximum functional contribution to the growth and developmental potentialities possessed by the child.

News and Notes

University of Illinois College of Dentistry

Applications are being accepted at the University of Illinois College of Dentistry for two dental research fellowships made available by the United States Public Health Service for the study of periodontal disease.

Complete information may be obtained from and applications directed to Dr. Isaac Schour, College of Dentistry, University of Illinois, 808 South Wood Street, Chicago 12.

In a recent study in Italy, conducted after the cessation of hostilities, Dr. Schour and Dr. Maury Massler noted that the Italian population suffered less from decay than the American population but more from periodontal disease (pyorrhea).

Whereas United States dental authorities have established an accepted index (DMF—decayed, missing, filled) for determining the extent of decay, no comparable index has been set up for determining gingival (gum) disease.

To work toward this end, the public health service, at Dr. Schour's suggestion, has granted $11,000 and established the two fellowships. Accepted applicants may also do part-time work toward a graduate degree.

Eight specialists from dental schools in Chicago will conduct a symposium on preventive dentistry November 25 and 26 as a climax to the University of Illinois' second 12-week refresher course in clinical and preclinical dentistry and wartime developments.

On November 25, Dr. A. O. Gruebbel, American Dental Association, Dr. L. S. Fosdick, Northwestern University, and Dr. R. G. Kesel and Dr. Maury Massler, University of Illinois, will discuss preventive dentistry from the viewpoint of public health, nutrition, biochemistry, and bacteriology.

On November 26, Dr. J. Blayney, University of Chicago, Dr. B. Orban, Loyola University, Dr. G. M. Teuscher, Northwestern University, and Dr. A. G. Brodie, University of Illinois, will consider caries, periodontal disease, orthodontics, and children's dentistry.

A similar symposium will be included in the third dental refresher course at the University of Illinois, beginning January 6. A similar roster of outstanding teachers and investigators will appear at that time.

Applications are now being accepted for the third course. They should be directed to Dr. Isaac Schour, College of Dentistry, University of Illinois, 808 South Wood Street, Chicago 12.

A maximum of 36 students can be accommodated. These courses were designed primarily but not exclusively for dentists returning to private practice after service in the armed forces.

American Association for the Advancement of Science

Dr. Isaac Schour, secretary, subsection on dentistry, American Association for the Advancement of Science, directs the attention of dentists to the symposium on "Biologic Aspects of Periodontal Disease," to be given December 29 in Boston as part of the association's annual meeting. All members of the profession are invited.

Officers of American Dental Association



Sterling V. Mead



H. B. Washburn

Dr. Sterling V. Mead, of Washington, D. C., is the new president of the American Dental Association. He was installed at the close of the 1946 meeting of the House of Delegates at Miami, Florida, succeeding Dr. Walter H. Scherer of Houston, Texas.

Dr. Mead was born Oct. 16, 1888, at Hutchinson, Kansas, where he attended the public schools and the Salt City Business College. He received his dentistry degree from George Washington University in 1914 and was awarded his bachelor's and master's degrees in science at Georgetown University. In 1929 he was president of the District of Columbia Dental Society, and, in 1932, served as vice-president of the American Dental Association. For several years, before being named president-elect in 1944, he served as chairman of the Association's committee on legislation. Dr. Mead has engaged in the practice of oral surgery in the nation's capital for many years.

Dr. H. B. Washburn, of St. Paul, Minnesota, is the new president-elect of the American Dental Association. He was elected by the House of Delegates at Miami, Florida, October 16, to succeed Dr. Sterling V. Mead, of Washington, D. C., who became the new president of the Association.

Dr. Washburn was born June 8, 1884, at Red Wing, Minnesota. He was graduated from the Dental School, Northwestern University, in 1905, and entered general practice in Zumbrota, Minnesota. In 1914 he went to St. Paul, where he is now engaged in the general practice of dentistry. He is a former president and secretary of the St. Paul District Dental Society and is a former president of the Minnesota State Dental Association. In 1926, he was chairman of the Full Denture Section of the American Dental Association. In 1940, he was elected a member of the Board of Trustees from the Tenth District and has served two terms.

Dr. Washburn will be installed as president of the American Dental Association at the close of its 1947 meeting in Boston, Massachusetts.



Harold Hillenbrand

Dr. Harold Hillenbrand, of Chicago, Illinois, new general secretary of the American Dental Association, took office following his election at the 87th annual meeting of the House of Delegates at Miami, Florida, in mid-October. Formerly, Dr. Hillenbrand was editor of the *Journal of the American Dental Association* and will continue to supervise the publication until his successor is chosen. As chief executive of the Association, Dr. Hillenbrand replaced Dr. Harry B. Pinney, also of Chicago, who retired after nineteen years of service.

The new dental executive is a graduate of the Chicago College of Dental Surgery, Dental School of Loyola University. He was engaged in the private practice of dentistry from 1930 to 1945 when he became editor-in-chief of Association publications. He formerly was editor of the *Illinois Dental Journal* and the *Fortnightly Review of the Chicago Dental Society*. He is also an associate professor of ethics and social relations at the Chicago College of Dental Surgery.

American Dental Association

Revised editions of the booklet, *The Story of an Association*, which describes the origin and development of the American Dental Association, are being distributed by the Bureau of Public Relations.

The new edition contains 26 chapter headings ranging from a short history of organized dentistry to a complete summary of current activities of the Association, its committees, councils, and bureaus. Included are outlines of the Association's governmental structures, a special chapter of finances showing major sources of income and principal expenditures, and descriptions of functions of each department. In all, the booklet's 64 pages provide a thorough view of professional activities of interest to all dentists.

Copies may be secured free of charge by sending a postal card request to the Bureau of Public Relations, American Dental Association, 222 East Superior Street, Chicago, Illinois.

American Dental Association

Dental schools in the United States graduated 2,666 new dentists during the 1945-1946 school year, the Council on Dental Education of the American Dental Association reports.

This total of new dental graduates is the second largest in recent history of the profession and reflects the accelerated training program adopted by dental colleges during the war years.

The wartime peak of dental graduates was reached in 1944-1945 when 3,212 dentists received diplomas.

During the five years prior to the war, the Council reported, the average number of dental graduates was only 1,712 per year.

Although the number of graduates increased during war years, enrollments of beginning dental students dropped sharply due to man-power demands of the Armed Forces. This decline in enrollment will result in smaller graduating classes during the next two to three years, the Council reported.

Present freshman enrollment at the nation's 40 dental schools, however, is in excess of 3,000 students, the highest in twenty years. Approximately 90 per cent of the beginning dental students are veterans of World War II.



Pioneer orthodontists on a hunting trip many years ago in Wyoming. From left to right, Pete Apel (guide); Dr. Harry Kelsey, Baltimore, Maryland; Dr. Albert H. Ketcham, Denver, Colorado; Dr. Frank Shelden, Kansas City, Missouri; and Dr. J. Love Young, New York City.

Registration at American Association of Orthodontists Meeting

At the 1946 meeting of the American Association of Orthodontists at Colorado Springs, Colorado, Sept. 30, Oct. 1, 2, and 3, the official registration including members, graduate students, exhibitors, essayists, and visitors amounted to 551.

It seems apparent, however, that many wives and other members of families of orthodontists did not register inasmuch as the Broadmoor Hotel registers 700 incident to the meeting.

Southwestern Society of Orthodontists

The Southwestern Society of Orthodontists will hold its twenty-sixth annual meeting, Feb. 2, 3, 4, and 5, 1947, at Hotel Adolphus, Dallas, Texas. All members of the American Association of Orthodontists are cordially invited. Nonmembers must be properly certified. JAMES O. BAILEY, Secretary, Hamilton Building, Wichita Falls, Texas.

Pacific Coast Society of Orthodontists

The Pacific Coast Society of Orthodontists will hold its next general meeting in San Francisco, California, Feb. 24, 25, and 26, 1947.

J. CAMP DEAN, 1624 Franklin Building, Oakland, Calif., President.

Northeastern Society of Orthodontists

The next meeting of the Northeastern Society of Orthodontists (formerly New York Society of Orthodontists) will be held at the Waldorf-Astoria Hotel, New York, on Monday and Tuesday, March 10 and 11, 1947.

District of Columbia Dental Society

The District of Columbia Dental Society sponsored a testimonial dinner in honor of Dr. Sterling V. Mead, President of the American Dental Association, on Wednesday, Dec. 4, 1946, at the Statler Hotel, Washington, D. C.

Notes of Interest

Dr. William P. Delafield announces the return from military service of Dr. W. Harrel Delafield and his association in the exclusive practice of orthodontics, Medical Arts Building, Dallas 1, Texas.

Dr. Harley G. Kushel announces the removal of his office to 507 Temple Building, Rochester, New York. Practice limited to orthodontics.

Dr. Philip Robin, practice limited to orthodontics, announces the association of Dr. Sidney Gerber for the practice of children's dentistry, Farragut Medical Building, Washington, D. C.

OFFICERS OF ORTHODONTIC SOCIETIES

The AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY is the official publication of the American Association of Orthodontists and the following component societies. The editorial board of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY is composed of a representative of each one of the component societies of the American Association of Orthodontists.

American Association of Orthodontists

President, Earl G. Jones - - - - - - - - - - - 185 East State St., Columbus, Ohio
President-Elect, Lowrie J. Porter - - - - - - - - - 41 East 57th St., New York, N. Y.
Vice-President, G. Vernon Fisk - - - - - - 818 Medical Arts Bldg., Toronto, Ont., Can.
Secretary-Treasurer, Max E. Ernst - - - - 1250 Lowry Medical Arts Bldg., St. Paul, Minn.

Central Section of the American Association of Orthodontists

President, C. S. Foster - - - - - - - - - - - - 803 Dows Bldg., Cedar Rapids, Iowa
Secretary-Treasurer, L. B. Higley - - - - - - - - 705 S. Summit St., Iowa City, Iowa

Great Lakes Society of Orthodontists

President, Willard A. Gray - - - - - - - - - - Medical Arts Bldg., Rochester, N. Y.
Secretary-Treasurer, C. Edward Martinek - - - - - - 661 Fisher Bldg., Detroit, Mich.

Northeastern Society of Orthodontists

President, Glenn H. Whitson - - - - - - - - - - - 80 Hanson Pl., Brooklyn, N. Y.
Secretary-Treasurer, Oscar Jacobson - - - - - - - - 35 W. 81st St., New York, N. Y.

Pacific Coast Society of Orthodontists

President, J. Camp Dean - - - - - - - - - - - - 1624 Franklin St., Oakland, Calif.
Secretary-Treasurer, Earl F. Lussier - - - - - - 450 Sutter St., San Francisco, Calif.

Rocky Mountain Society of Orthodontists

President, Henry F. Hoffman - - - - - - - - - - 700 Majestic Bldg., Denver, Colo.
Secretary-Treasurer, George H. Siersma - - - - - - 1232 Republic Bldg., Denver, Colo.

Southern Society of Orthodontists

President, J. E. Brown - - - - - - - - - Merchants National Bank Bldg., Mobile, Ala.
Secretary-Treasurer, Leland T. Daniel - - - - - - 407-8 Exchange Bldg., Orlando, Fla.

Southwestern Society of Orthodontists

President, Brooks Bell - - - - - - - - - - - - Medical Arts Bldg., Dallas, Texas
Secretary-Treasurer, James O. Bailey - - - - - - Hamilton Bldg., Wichita Falls, Texas

American Board of Orthodontics

President, Oliver W. White - - - - - - - - 213 David Whitney Bldg., Detroit, Mich.
Vice-President, Joseph D. Eby - - - - - - - - - - 121 E. 60th St., New York, N. Y.
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James D. McCoy - - - - - - - - 3839 Wilshire Blvd., Los Angeles, Calif.
Stephen C. Hopkins - - - - - - - 1726 Eye St., N.W., Washington, D. C.
Reuben E. Olson - - - - - - - - - 1115 River Blvd., Wichita, Kan.

In the January issue each year, the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY will publish a list of all of the orthodontic societies in the world of which it has any record. In addition to this, it will publish the names and addresses of the officers of such societies.

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David Weisberger

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Original Articles

THE CLINICAL STATUS OF THE MANDIBULAR THIRD MOLAR

COMDR. HARRY L. LEVIN, (DC) USNR

It is the traffic of ideas that has the greater
value to humanity than the traffic in silks and jade.

INTRODUCTION

NOT since the amalgam war has the dental profession been rocked by the controversial issue of the mandibular third molar, namely, whether or not to extract this member in all cases regardless of the condition of its retaining periodontium after its eruption. Some have gone so far as to extract the crown of this tooth in its varying stages of structural formation. For these operators thought and still think that curettage of the root bud, after a liberal flap was thrown back, caused the remnants of this specialized tissue and all the tissues connected therewith to slough out, with a rapid recovery as a result in most cases where aseptic surgical procedure had been adhered to. By this method it can be seen any trouble this member might have caused in the future has thus been checked, and any usefulness it might have served been eliminated. But it must not be overlooked that all elective surgery may be dangerous to life and may be followed by distressing effect.

This author does not advocate the indiscriminate extraction of all third mandibular molar teeth regardless of its pathology, for after three years of careful study of 1,031 case histories while serving aboard the U.S.S. Pocomoke, it has been found definitely that in 65 per cent of these patients the tooth in question was serviceable and that there was no pathology present nor a history of any previous disorders. The presence of a pericoronitis is one of the most important single factors causing loss of man hours, fighting inefficiency, and other possible complications. It is advisable in these cases to remove the tooth if condemned, when safe to do so, regardless of the anatomy and malposition of the tooth. It is a good policy to leave alone a deeply impacted inactive tooth when there is no present disorder or history of any previous disturbance arising from the tooth.

Bourgoyne[1] states, "I believe that the lower third molars should be removed within one year after eruption of the second molars, or about thirteen years of age." The thought behind this statement is too one-sided and must be considered as coming from the pen of an exodontist.

Delivered before a joint meeting of Naval, Medical, and Dental Officers of the Naval Air Training Center at Chase Field, Beeville, Texas, in March, 1944.

The opinions or assertions herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

Remove the offending tooth when conservative and therapeutic measures have failed. If the exigencies of the service do not allow for conservative treatment, then removal is in good order since conservative treatment does not assure complete recovery to the normal when circumstances involving this tooth are not normal. It stands to reason that where the oral hygiene is going to be at a low ebb and conditions are always rife for recurrence of an acute condition, this tooth should be extracted. Under normal conditions it is most difficult to keep this area of low oxygen tension clean. The ever-present parasites need very little stimulation in form of irritants and at those times when there are no toothbrushes which will satisfactorily clean this area, removal is justifiable.

In the field and on ships without proper means for dental treatment, such as minesweeps, destroyers, submarines, and small auxiliary craft, it is advisable to extract when the history is that of intermittent attacks with possible future recurrences. This does not imply indiscriminate and wholesale extractions of all third molars, good and bad. In many instances this tooth may prove useful as an abutment for a bridge or in some cases the tooth will move in to take the position of an extracted second molar. As a molar it can be considered, if properly occluding with its opposing member, a means to future promotion and retention in the service which otherwise would not be the case were these extracted.

Many operators, it can be shown, refuse to fill these teeth, just as many in years gone by refused to fill deciduous teeth, harboring the illusion that in a short time they would be cast off, hence there was no need to do battle with the obstreperous child. This is a vicious attitude, and if dentistry is to be considered a profession and a science, every effort should be made to preserve this tooth and every chance given for its survival.

When the pressure resulting from growing third molars causes excessive wear on the contact points of adjacent teeth and thus causes abrasion on these surfaces, the third molar being impacted, then it is permissible to extract the offending tooth.

Though there be no pathology or lack of symptoms, one of the plausible reasons for the removal of this tooth is for prosthetic requirements. The removal of the unerupted third mandibular molar tooth may be considered in good order so that a patient may have uninterrupted usefulness of full dentures or better partial dentures.

ANTHROPOLOGY

Evidence of man upon the face of this earth has been computed as far back as approximately one million years. The antiquity of man is a matter of interest both to paleontologist and allied scientists as well as the laity alike. Our knowledge of man, extending back thousands of years antedating the birth of Christ, is based solely upon the findings of fossil bones in the various parts of the earth, of which the bones of the skull play no little part. In 1856 a human skull of an unusual type was found in the Neanderthal Valley in Germany. Experts who examined the specimen declared that it belonged to a new and particular form of the genus *Homo* and gave the name "Neanderthal" to the new species, meaning Neanderthal Valley man.

Later explorations have more than confirmed the opinions of anthropologists, who detected in these stocky little men a most important connecting link between the past and present in human evolution. From what we are able to gather by measurements of the completed skulls from fragments and fossil bones, the Neaderthal men were short men with thick necks, big hands and feet, and legs quite short below the knees. The forehead was low, the face and jaw projecting forward, and ridges over the eyes were most pronounced. All in all the Neanderthal man was a most unprepossessing little fellow, but abundantly able to look after himself in the cold, wet, and dreary climate of his time.[2]

From an anthropologic viewpoint, it has been definitely shown that the skull of the Java man *(Pithecanthropus erectus)*, the most famous of early men, was of such large proportions that many anthropologists are not in accord as to whether it was the skull of man or breast. There are many other skulls of the lesser known to the laity which helped in the restorations of what approximated primitive man. The Mousterian relics, the Galley Hill man, the Ipswich man, the Tilbury skull, the Piltdown, Heidelberg, Peking, Rhodesian, and Cro-Magnon men are just a few of the many thousands of skulls and fossil bones unearthed. Significantly enough, in their reconstructed skulls all show sockets for three molar teeth in either side of the maxillary and mandibular jaws. It therefore was not a question of imaginative reconstruction. It was positive fact.

These prehistoric men lived either during the early Pleistocene or some remote time in the later Glacial Age. One must bear in mind their jaws were massive and well developed, square type predominating. The abraded surfaces of the mandibular third molars together with sufficient room in the dental arch, it can be seen, served as a useful organ of mastication. Unfortunately, this cannot be said of all the molar teeth of man today, of which more will be said later in this paper. The evolution of this tooth is synonymous with the evolution of man in general. It would be wise to trace back to that time when it served as a useful member this tooth whose usefulness is being dissipated until the time will come when it will no longer function as it was originally intended. Poor adaptation to its environment has led to the pathogenicity which will eventually lead to its own extinction.

By hypothesis it may be assumed that there is some connecting link between man and the Anthropoidea of the higher order, which include the gorilla, pseudogorilla, chimpanzees, orangutan, gibbons, and their suborders and their closely related members. The classification of their teeth is identical with that of man today and those of the primitive types. The classification is herewith given:

Man (today): $I - \frac{2-2}{2-2}, C - \frac{1-1}{1-1}, Pm - \frac{2-2}{2-2}, M - \frac{3-3}{3-3}, - 32.$

Primitive Man: $I - \frac{2-2}{2-2}, C - \frac{1-1}{1-1}, Pm - \frac{2-2}{2-2}, M - \frac{3-3}{3-3}, - 32.$

Anthropodia: $I - \frac{2-2}{2-2}, C - \frac{1-1}{1-1}, Pm - \frac{2-2}{2-2}, M - \frac{3-3}{3-3}, - 32.$

(I = Incisors; C = Canines; Pm = Premolars; M = Molars.)

The only difference seems to be in the shape of the canines of the primates. The tooth is markedly pronounced and much longer than those of primitive man and his progeny, the man of today. Primitive man had also pronounced canines, but not nearly so large nor so long. See illustration (Fig. 1) comparing the Heidelberg jaw with the jaw of the higher Anthropoidea and of the modern man with the third mandibular molar teeth intact and in the same position.



Fig. 1.—The Heidelberg jaw compared with the jaw of the higher Anthropoidea, chimpanzee, and of a modern man. (Courtesy of the American Museum of Natural History, New York.)

It is quite apparent during the early stages in the evolution of the earth prior to the Carboniferous Age that plant life was abundant, so much so the animals and man as we knew them were of enormous size. Whether the interplay of oxygen and carbon dioxide had anything to do with it cannot be clearly shown. Lizards 20 and 30 feet long, other animals proportionately as large, prevailed; some flew, others jumped, and most bounded through the jungles of that time. Prehistoric man had proved himself able to cope with the amazing turn in events as the Glacial periods set in. He survived, the rest did not. Something happened, the plant life on the earth bogged down and withered, and so these enormous creatures have long since become extinct. The first Glacial age came on. As though this was not sufficient to cover the earth with huge mountains of ice, a second came, then a third and even a fourth. Man the primitive lived and died through these epochs from the earliest Paleolithic ages on past these Glacial epochs.

There is proof that prehistoric man may have had more than the full complement of teeth in terms of the dentition as we know it today. There are also grounds for contesting a difference in the number of anterior teeth, particularly the incisors, but not so with the molars. As may sometimes happen, the mandibular third molar teeth may be congenitally absent and by the same token a fourth molar tooth may be present. The same may be said of the congenital absence of one or more of any tooth in the head. Be that as it may, the pathologic and neurologic symptoms make this an adult problem affecting the health and efficiency of the serviceman today.

ANATOMY

The mandibular third molar is an anomaly. Its position in the jaw is unpredictable, as are its root formations and numbers. Unlike its adjacent molars

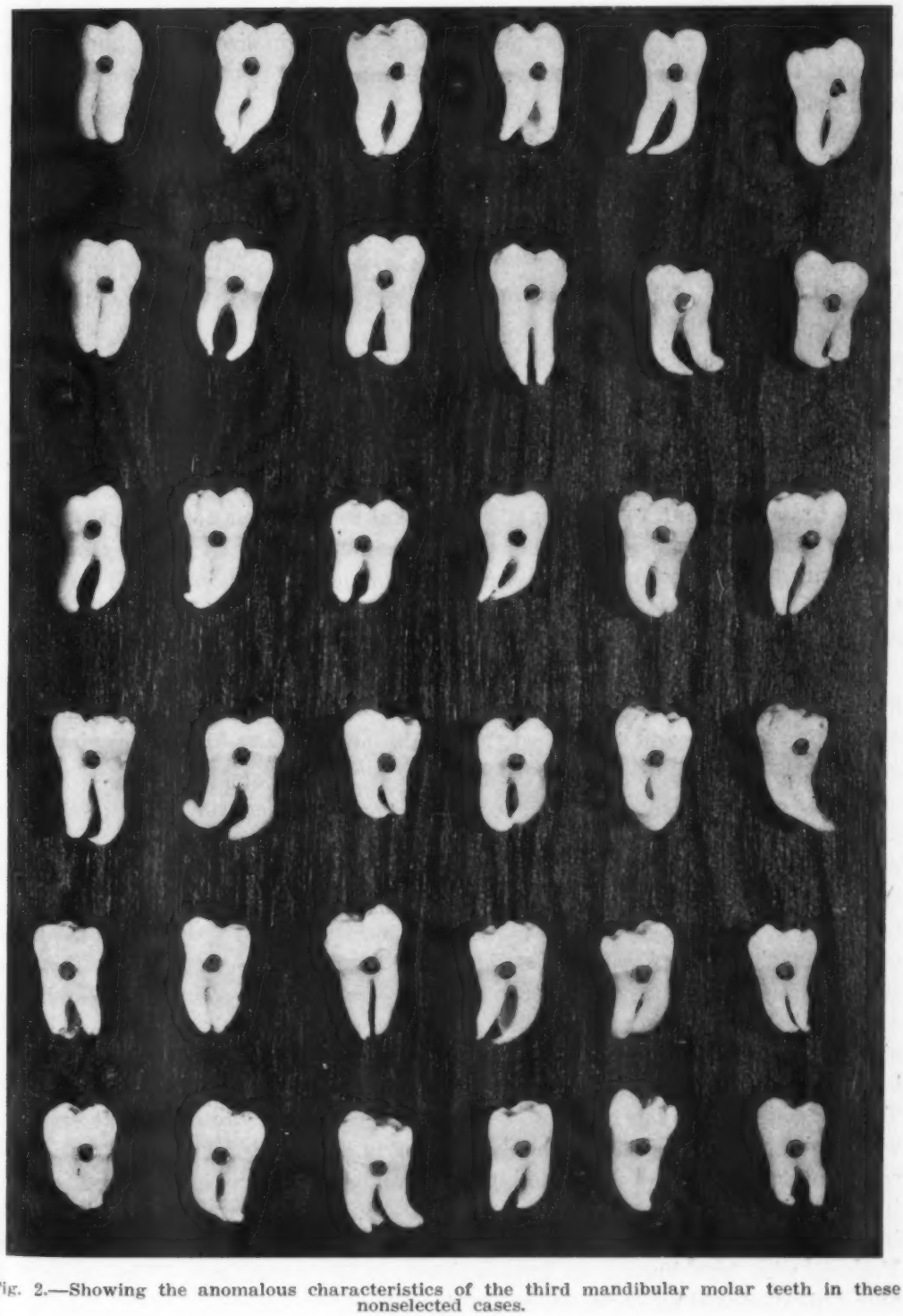


Fig. 2.—Showing the anomalous characteristics of the third mandibular molar teeth in these nonselected cases.

which always have two roots, their roots, however, might be fused and conical with a decided distal curvature. This tooth cannot be described adequately to apply to every third mandibular molar. It varies so, in size, shape, and number of roots, the contour of its surfaces, and the number of cusps on the crown. The roots may present fingerlike projections, very much like improperly grown carrots, while in other cases they may be gnarled and twisted and like as not the apices will present feet pointing distally. At times the roots may be separated with a dense bone formation between, making removal a difficult procedure. (See Fig. 2.)

The tooth varies considerably in its relation to the jaw. The crown may tilt forward, may be everted inward or in an outward position. It may erupt in a normal manner or be partially covered by the muscular folds of the buccinator and pterygoid muscles, and so impinge upon its normal eruption. Current literature paints a very dismal picture for this tooth and goes far in predicting its eventual relegation to that of a vestigial organ. Reviewing clinical findings may possibly throw some light on these indiscriminate attacks, going so far as to condemn even the healthy and serviceable member as well. The same arguments have been put forth about the removal of the vermiform appendix, and in due time wholesale removal has proven fallacious and, as in every operative procedure, fraught with dire consequences, for no matter how simple the operation and aseptic the field infection sometimes develops, jeopardizing even life itself.

It may then be asked what has happened to this tooth, which at one time must have served as a useful healthy member, to have caused it to assume its anomalous position and characteristics. Why has it become the *bête noire* of the oral cavity?

It is quite evident that the contour of the skull and the bones of the face have much to do with the fullness of the dental arch. It may be presumed that a broad face or even a full round face would be less apt to cause crowding of the teeth than a narrow, rodent, or tapering type of jaw. However, this may not always be the case, as impacted teeth are very frequently observed in the mouths of Filipinos, American Indians, and those of the Negroid races. Nodine[3] stated that Professor Fowler[4] sixty years ago claimed that "defective conditions of the third molars (impacted third molars) are no monopoly of the most highly civilized races, but may be found among the most abject and degraded of the human species: Negroes, Melanesians, Australian Aborigines, and Tasmanians." To refute those who have been so bold as to have written volumes on the modern way of life, especially that which had to do with the habits of eating processed, soft, and concentrated foods and their effect on the human dentition, careful investigation would have shown that the oldest human specimen showing an impacted tooth was the Mousterian man, a prehistoric man unearthed in Vézère, France, who was known to have existed 40,000 years ago. The unearthed mandible disclosed an impacted canine tooth.[5]

Neither are impacted teeth exclusively a man's debility. It is found in animals as well, and what is more interesting, it can definitely be shown to have



Fig. 3.—Impacted molar from pathologic mandible of smilodon (saber tooth cat). (Courtesy of Los Angeles County Museum.)



Fig. 4.—Enlarged detail of nonerupted premolar from pathologic mandible of smilodon. (Courtesy of Los Angeles County Museum.)

been due to a maladjustment in a jaw of a prehistoric beast; a sabre-toothed tiger of the Pleistocene Age, whose estimated antiquity is 75,000 years, is shown to have what would correspond to an impacted third mandibular molar tooth.[6]

That the reader might not feel this demonstration (Fig. 3) of an impaction in a prehistoric animal is unique, further photographic evidence is herewith submitted in Fig. 4 to show that impactions during this epoch of the earth's evolution must not have been unusual.

ETIOLOGY

Many theories of the past and present have been advanced to offer an explanation for the cause of this malformation, but none substantiated by conclusive proof. However, it might prove interesting to review a few of the most popular theories propounded over a long span of years.

John Hunter in his *Natural History of the Teeth,* published in 1771, and Charles Darwin in his *Descent of Man,* 1871, were of the opinion that the teeth and jaws of prehistoric man and his progenies differ in size and possibly in shape. During the process of evolution the tendency was that the teeth and jaws did not keep pace with each other in their growth. It was not uniform and equal; the jaw grew disproportionately smaller than the teeth they contained. There was also a disparity of the teeth and the palate.

Factors of a pathologic nature in the human make-up are known to have disturbing influences and a definite bearing on eruption of teeth. Endocrine dysfunction with its chemical mechanism affects both the deciduous as well as the permanent set. The thyroid glands have an influence on deciduous teeth directly and the permanent indirectly.

Hyperthyroidism would have a tendency to overstimulate the density of bone formation and thus cause the bones to become excessively large. The jaws become heavy and unduly massive, which certainly does affect erupting teeth. The dense jaws cause the third mandibular molar tooth a great deal of difficulty during its eruptive processes. On the other hand, hypothyroidism causes undevelopment and delayed eruption of the teeth and in many instances total absence of one or more.

There are many other factors, to be sure, many of an embryonic nature, which have to do with the interference of the normal growth of the tooth bud. Some investigators theorize that the failure of the mandibular third molar tooth to rotate from an oblique lingual and mesial position to a vertical and occlusal position prevents this tooth from emerging from the jaw.[7]

There is a theory which attributes the abnormalities in position of erupting third mandibular molar teeth to the frequent defective intrauterine development, due to syphilis, tuberculosis, and malnutrition.

Pterygoid mandibular ligament disorders with resultant ankylosis in early life have definite bearing on the erupting third mandibular molar tooth, since it limits the space for its eruption.

Robert Bunon, in 1743, stated, "Rickets is one of the chief causes of the lateness in the eruption of deciduous teeth, irregularities, overcrowding, and malformation of the jaws." Deficiency of vitamin D causes imperfectly calcified

bones and the normal process transition from the deciduous to the permanent teeth is upset.

Cysts, odontomas, and other new growths, benign and malignant, may divert teeth from their normal path of eruption or prevent them from erupting at all. Osteomyelitis in the jaws of the young affect normal eruption of the teeth that are to follow.

Muscular attachments, heretofore described as the buccinator and ptyergoid muscles, partially or fully overriding the crowns, accentuate the gingival pockets. Much calculus, materia alba (soft white deposits composed of food debris, mucus, dead epithelial cells, purulent matter, and various types of bacteria), and inspissated saliva will collect therein with later bacterial infiltration to help break down the normal condition around this tooth, thus setting up pathologic processes. It is also evident in mouths showing a calcium and phosphate deficiency that calculus accumulations are heaviest on the internal surfaces of the lower anterior teeth, as well as the third mandibular molar teeth. The inorganic composition of this tooth is usually poor in these cases, making it susceptible to extensive caries. Furthermore, any attempt at reconstructive conservation indicates a poor risk, for decay will reoccur where the filling that has been placed ends and the poorly constituted enamel begins. Unlike the other teeth, it cannot draw the requisite inorganic elements from a source, as does the fetus from its mother, for at the end of the sixth year the tooth bud is completely formed and prepared for its transit by slow stages to its ultimate site in the jaw.

In his recent enlightening work Nodine[3] concludes that the primary factor responsible for tooth impactions is the redundancy of the epithelial organ and consequent development of the organ beyond its allotted area.

Cryer[9] is also of the opinion that the angulation of the mandible seems to have some bearing on the normal arrangement of the teeth. Foreshortened jaws cause a diminishing of the space allotted for erupting third molar teeth and in all likelihood are the cause of impactions in the coronoid process.

Mental development seems to be closely associated with erupting teeth and delayed eruption. Brain and teeth are both derived from ectodermic tissue and are thus liable to the same disturbances.[1]

Factors of an exogenous nature are definitely known to influence the alignment of teeth, causing them to assume an irregular position and crowding in the alveolar arch. Crossbreeding does not always bring out the best in man. Since man is not controlled in the selection of his mate, and since opposites attract each other, the results are not always advantageous from a eugenic point of view.

Thumb-sucking and lip- and tongue-biting in the young do not make for normal occlusion, nor the normal in tooth arrangement, which might ultimately affect the eruption of the third mandibular molar tooth.

CLINICAL FINDINGS

During the latter part of 1941, while serving aboard the U.S.S. Pocomoke, a small craft tender stationed at Argentia, Newfoundland, I observed for the first time that manifestations of oral disturbances, other than carious teeth, were

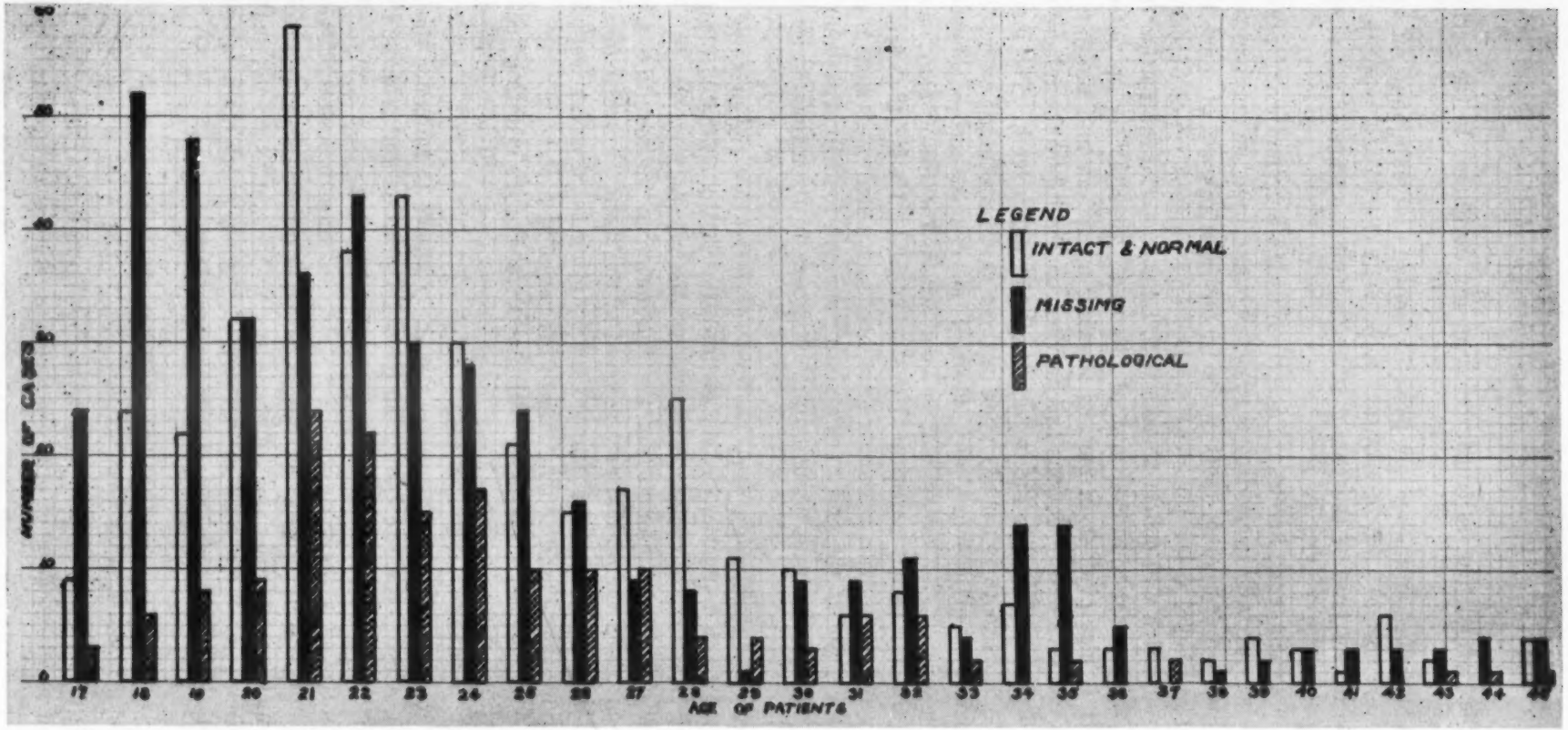


Fig. 5.—A comparative study of the third mandibular molar tooth, left side, in 1,031 cases, showing the normal and intact, pathologic, and those teeth which were missing from their allotted space in the mandible.

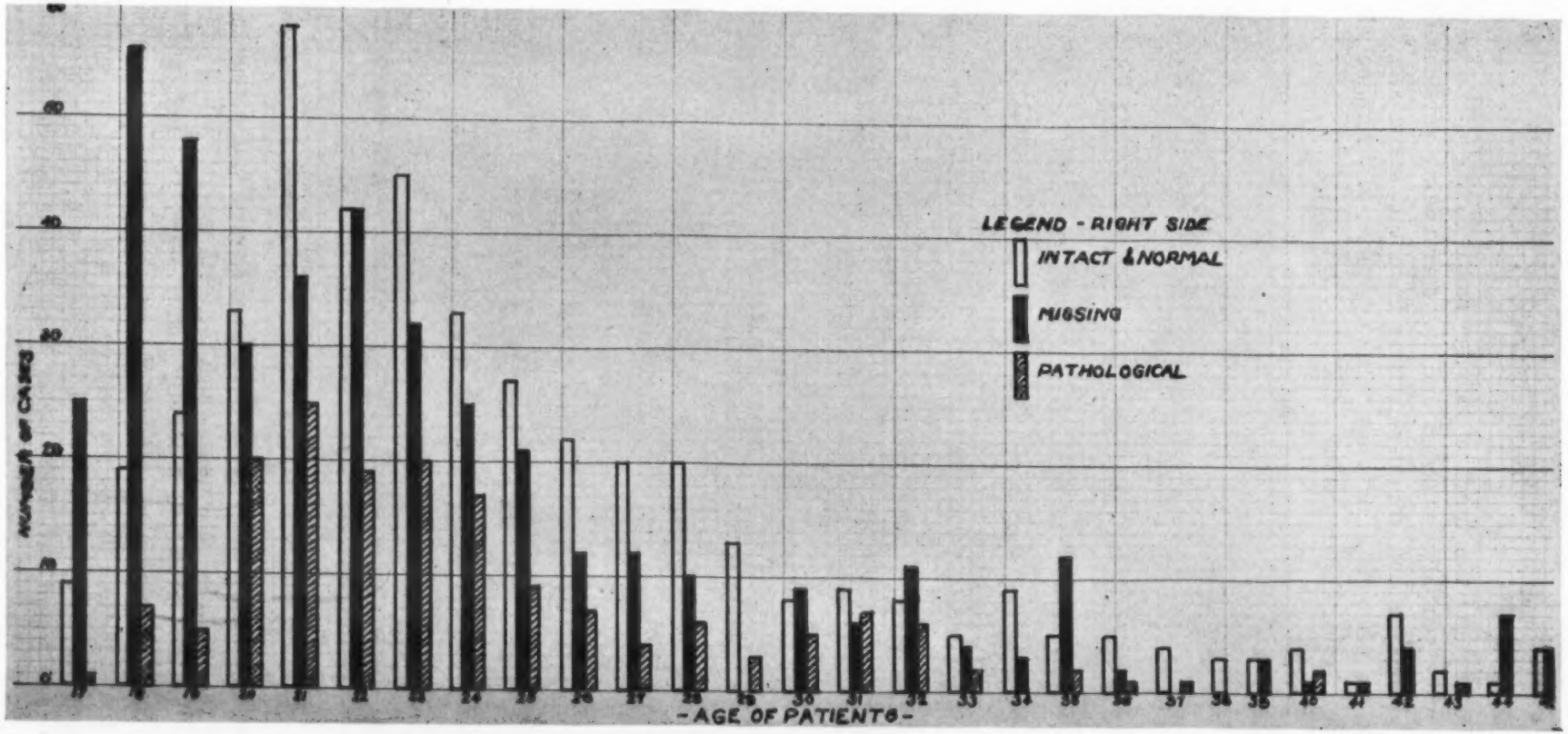


Fig. 6.—A comparative study of the third mandibular molar tooth in 1,031 cases.

usually worse around the third mandibular molar teeth, especially so when these teeth were partially impacted and the surrounding periodontium was pathologic. Knowing how disorders arising from this source can affect the efficiency of the personnel, this study was undertaken to determine what percentage of our Armed Forces was involved.

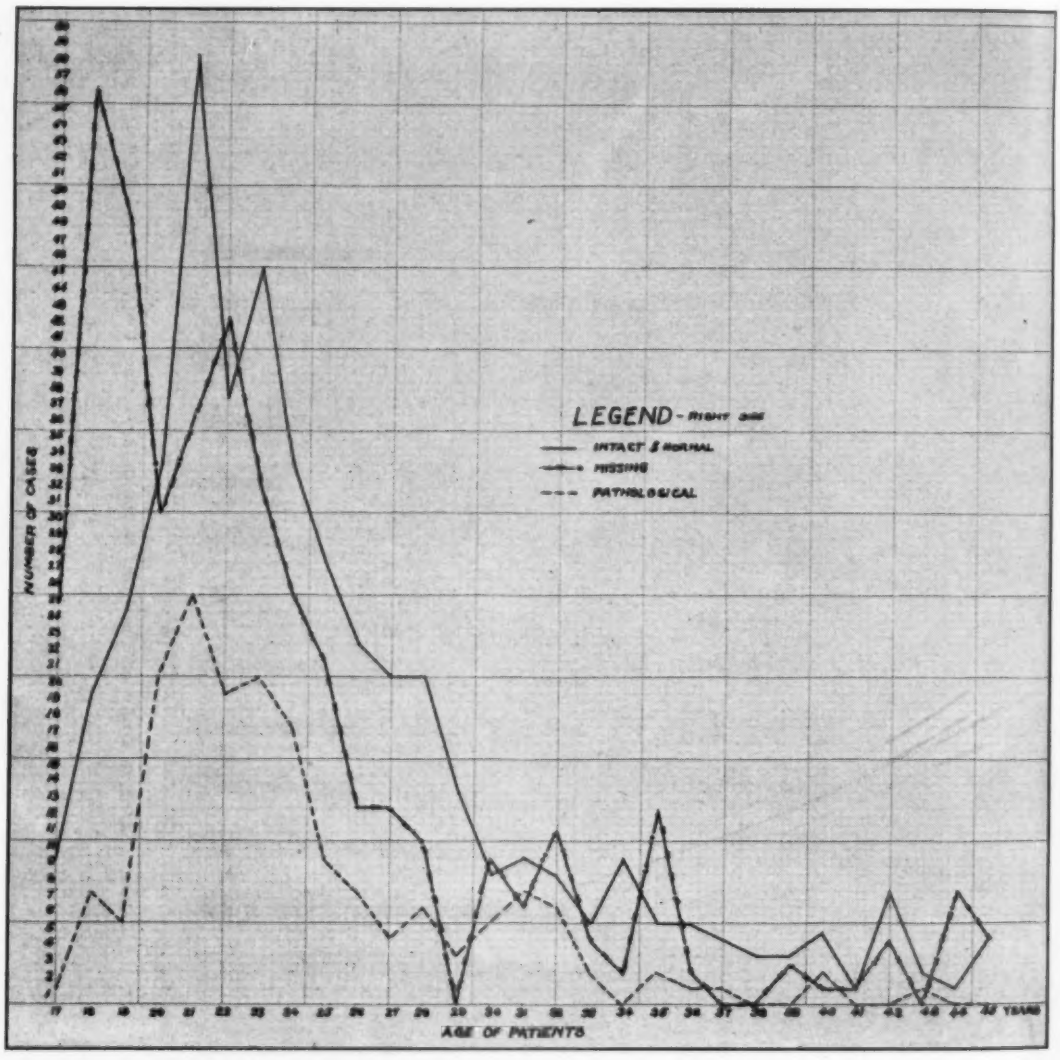


Fig. 7.—Graphically representing the trend of the intact and normal, missing and pathologic, third mandibular molar tooth in 1,031 cases.

Clinical findings in 1,031 nonselected cases showed that pathology existed in 17.6 per cent of those patients in which the tooth was still intact on the right side. The left side presented a slightly higher ratio, 18.2 per cent, but this difference is mere coincidence. After the supposed period of eruption, the tooth was found missing in 24.3 per cent on the right side of the mandible and 28.4 per cent on the left side. It can be readily seen that the percentage of pathologic cases would have been higher were it definitely known within this

age group of eruption whether the tooth had fully erupted and was removed for pathologic reasons between the seventeenth and twenty-fifth years. However, it must be presumed that some third mandibular molars will erupt before the seventeenth year while others after the twenty-fifth. Interrogation of the patients as to the reason for the missing tooth in many cases brought forth a history of pathology which accounted for its removal. Just what the exact

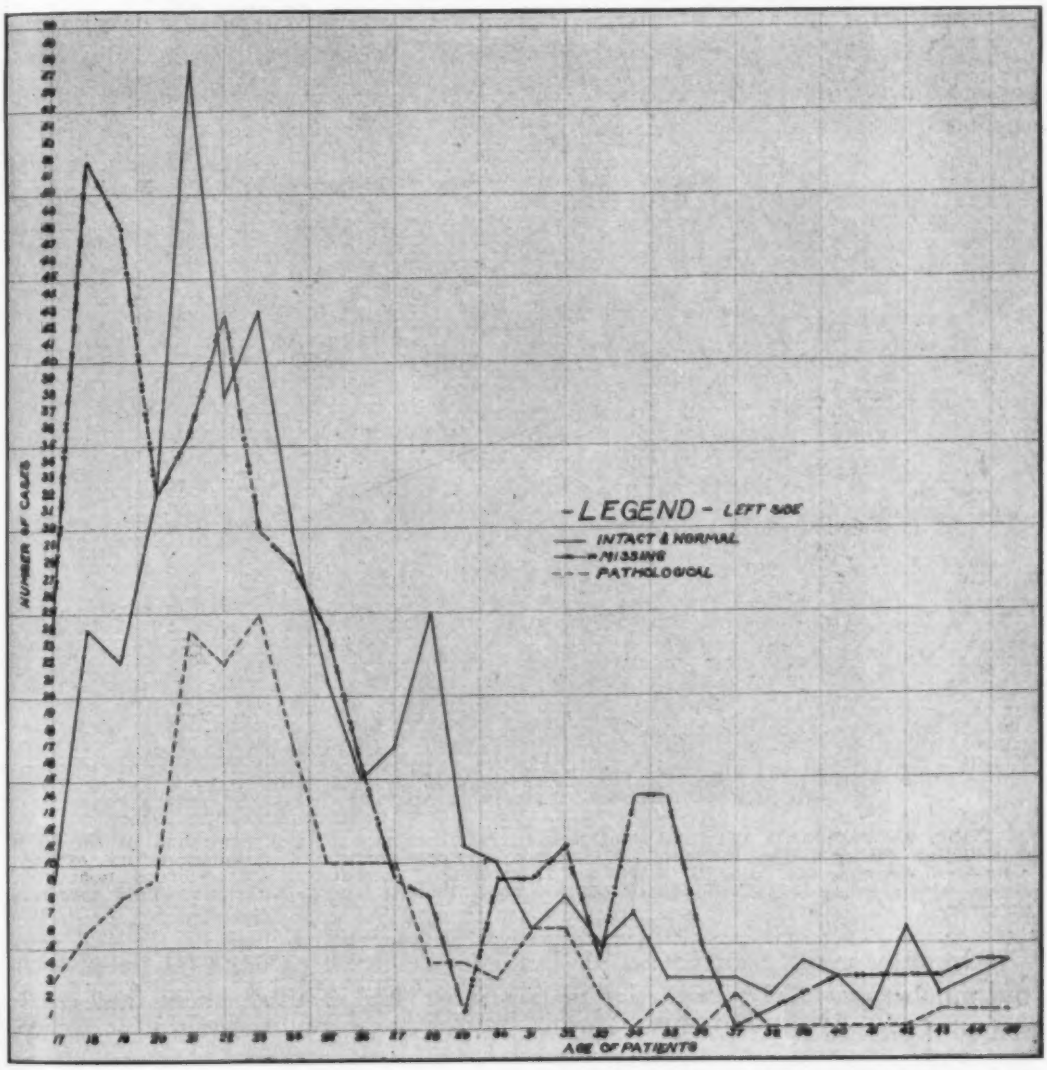


Fig. 8.—Graphically representing the trend of the third mandibular molar teeth which were intact and normal, missing and pathologic, in 1,031 cases.

percentage was could not be truthfully ascertained, since many patients could not remember ever having any trouble or having them removed at all, even though x-ray findings showed definite removal in a large percentage of these questionable cases.

Bourgoyne,[1] in a careful investigation, found that the third mandibular molars were congenitally absent in 5 per cent of three hundred case histories

of physicians intelligent enough to give exacting information. My findings are in accord with the findings of Bourgoyne; from the study of 1,031 case histories I was able to judge that 5 per cent would be a fairly accurate figure.

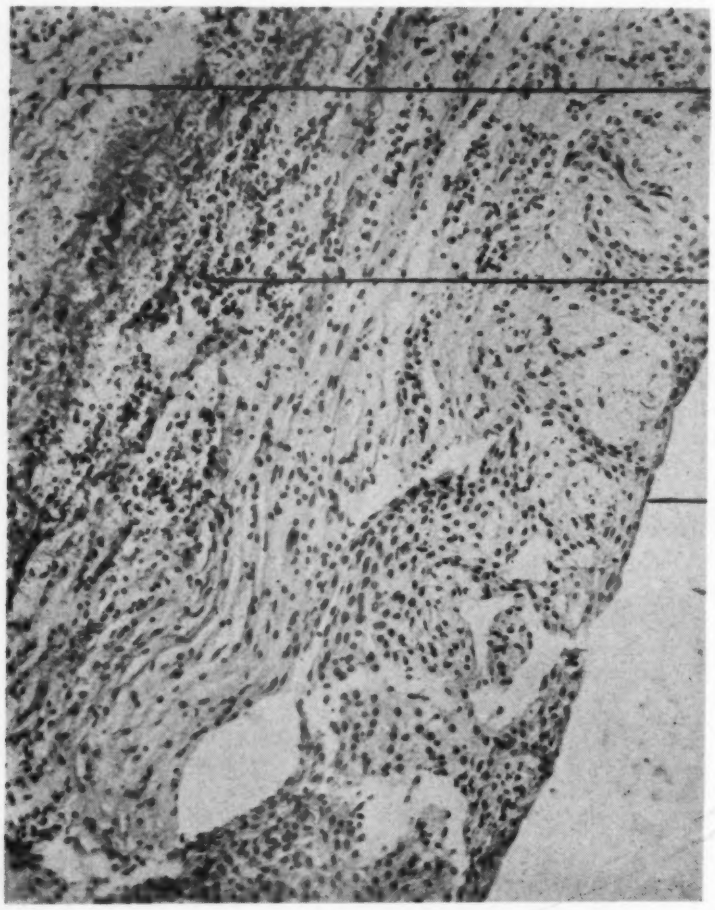


Fig. 9.—Showing a potential dentigerous cystic remnant of the tooth bud of the third mandibular molar tooth, infected. *X*, Subepithelial accumulation of inflammatory exudate cells—lymphocytes and plasma cells. *P*, Hypertrophy epithelium. *CT*, Fibrous connective tissue which encapsulated the roots of the third mandibular molar tooth. This tissue is sclerotic.

In the careful examination of these one thousand patients tabulated as to the number of third mandibular molars intact and healthy, those that represented a picture of pathology and those that were considered missing are hereby graphically presented. The right side was charted separately from that of the left. Significantly enough, there is not much difference in the graphic picture as shown in the chart on the right side and that represented by the chart of the left side. Both sides follow very much the same pattern in all three instances in those that were intact, those that were pathologic, and those considered missing. The ages of 21 and 22 years may be considered the period of the greatest incident in which this tooth is found most frequently healthy, and also that time when the incidence of pathology is greatest. After the thirtieth year the tooth is seen in ever-diminishing numbers, until at 45 years of age it is seen only occasionally.

PATHOLOGY

No other tooth when extracted for any reason will bring along its encapsulation remnant of the tooth bud from which it grew as may sometimes happen in this region. This capsular tissue when inflamed and pathologic must be removed entirely or else cystic formations may develop later (Figs. 9 and 10).

Microscopic section of this tissue, when pathologic and of a chronic inflammatous variety, shows a regular stratified squamous covering epithelium which is partly ulcerated in areas. The epithelial cells are slightly hyterplastic in other areas but show no neoplastic change. The submucosa shows granulomatous areas having many infiltrating plasma and lymphoid cells. There are, however, no neoplastic or specific inflammatory cells to be seen.

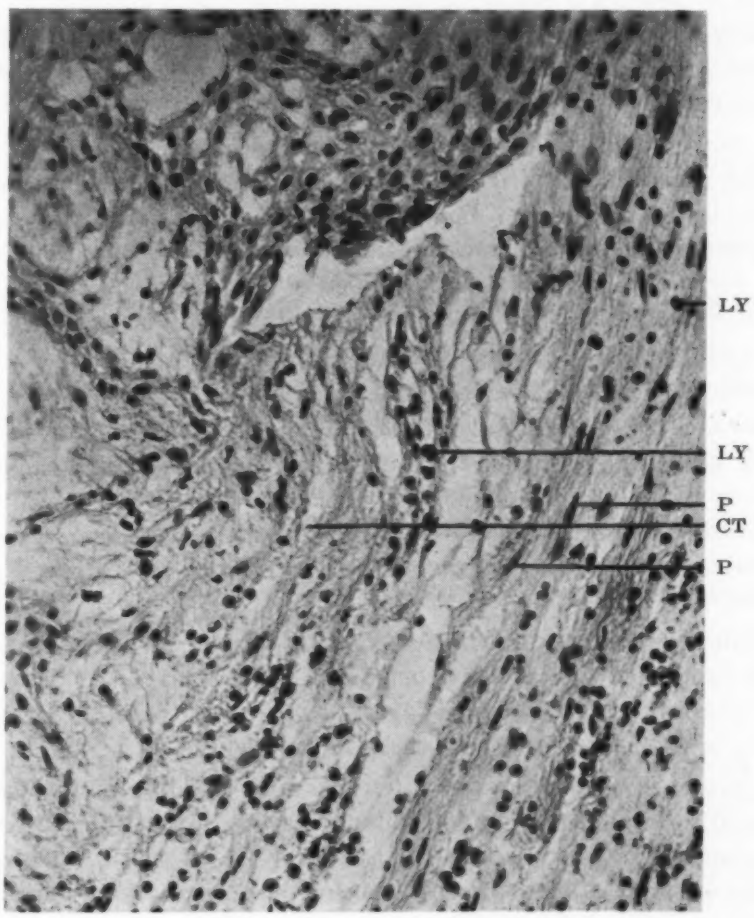


Fig. 10.—Higher magnification of the remnant of tooth bud with infiltration of the connective tissue (*CT*) with lymphocytes (*LY*) and plasma cells (*P*). Evidence of adamantine epithelium is absent.

It is not unusual to have an exacerbation of the inflammatory process on the lower right side, with all its unpleasant sequelae, while the left remains quiescent, and at some future date to have the left side flare up as did the right, the right side remaining in a state of mild chronic infection. Again, the right mandibular third molar may come through perfectly normal while its opposite

member erupts in any position save that which would be considered normal, or vice versa. Its growth and reactions, therefore, cannot be considered bilateral.

Though many of these teeth are partially or fully impacted, they may make their presence known as an acute infection, which may later assume a chronic course. "Subsidence of the attack does not mean the elimination of the infection, rather that the disease has become chronic with a tendency to acute exacerbation, at some future time when vital powers are lowered."[14] The sore throat, trismus, the swelling of the regional lymph nodes, the malaise and fever are certainly suggestive of an acute condition. It will be noted that though there be a period of much uneasiness with the already mentioned syndromes, with the elimination of the course of the acute condition complete recovery may be expected. If treatment of a conservative nature is instigated, there will be a subsidence of the acute condition into one of latency; however, there is always a possibility of a later reawakening to activity and occasional clinical symptoms unless operative procedure is undertaken toward total elimination of the causative agent, which in our case is the impacted tooth.

One must be fully cognizant of the lowered vitality of the tissue in and around the area of the third mandibular molar, and subsequent operations are always fraught with danger though technique and asepsis may apparently be above reproach. Therefore, an upset equilibrium between the host and parasite will call forth recognizable reactions should the parasite gain the upper hand. It is also obvious that, in unhygienic mouths, Schmutz pyorrhea as well as Vincent's gingivitis add to the existing pathology of the circumferential tissues of the tooth in question.

Sometimes as an untoward result from the extraction of teeth, jaws are fractured, principally during the removal of impacted third molar teeth. As pointed out by Ivy and Curtis,[12] this has happened in 5 per cent of the cases referred to them over a period of three years. It can be seen, therefore, it does happen often enough to warrant our utmost care in any surgery performed in this region.

Pathologic fractures are also seen occasionally, but these arise from complicating tumors, cysts, and osteomyelitis which may or may not have their origin around the third mandibular molar teeth but are rather in themselves extensions of pathologic processes that arise elsewhere.

The presence of a congestive gingivitis with bleeding and swollen gum flaps over partially erupted third mandibular molar teeth that fails to respond to conservative and therapeutic measures toward its eradication or toward localization of the inflammatory processes, is suspicious of an avitaminosis, niacin, riboflavin, and ascorbic deficiency.[16]

Were it possible to overcome the objections of those who advocate extraction in every case and those operators who do not consider it worth while and necessary to place fillings in carious third molars, then we will have come a long way in wiping out the contempt that is held for this tooth. One must not lose sight of the fact that at least 65 per cent of third molars can be considered normal and, as such, might be presumed to serve a useful purpose.

DIAGNOSIS

Fortunately diagnosis of the inflammatory disturbances arising from impacted third mandibular molar teeth is not difficult. The exposed cusps and resultant pericoronitis, together with slight trismus due to edema and a dull throbbing pain, are significant. The pain is seldom, if ever, of the excruciating variety, but rather one of fullness, arising from the inner angle of the ramus. Sometimes it may manifest itself as a radiating pain arising from the front of the ear. There is little rise in temperature and the pulse rate will vary with the severity of the case. The patient may complain of malaise and even slight chills when there is an infiltration into the areolar cellular tissue of fluid products of infection. Usually a history of previous intermittent attacks, together with the necessary roentgenograms which disclose the bony involvement and the malposition of the tooth, is conclusive evidence of third molar involvement. Free pus might be shown by gentle penetration with a sterile probe into the abscess which may form within the surrounding tissues of the tooth involved. Further points may be taken up by the summation that follows.

1. Local symptoms:
   a. Inflammation and congestion.
   b. Pain in the immediate area; palpation of the tooth by percussion brings forth sharp paroxysms of pain.
   c. There is a definite interference with mastication due to edema and partial trismus.
   d. The pressure of the upper jaw upon the swollen tissue of the lower may be reason for extraction of the maxillary occluding tooth before the removal of the third mandibular molar tooth.
2. Systemic symptoms:
   a. General malaise.
   b. Toxemia.
   c. Temperature slightly raised.
   d. Arthritis may result as a consequence (foci of infection).
   e. Neuralgia, referred pain, earache, sore throat from extension downward of the inflammatory processes.
   f. Vincent's angina.
   g. Adenitis.

TECHNIQUE OF REMOVAL

It is necessary to take good intraoral and extraoral roentgen-ray pictures prior to the operation, preferably long enough before to allow for the drying of the films. Trying to visualize the exact position and shape of the roots leads to pitfalls, which at times prove very embarrassing to the surgeon and cause much discomfort to the patient.

Never take the extraction lightly, but prepare for the most resisting efforts on the part of the tooth to be dislodged. Otherwise the employment of instruments not properly sterilized will usually lead to much grief. It is best not to have to use them, but to be prepared, knowing that if an emergency arises the instruments are there sterile and ready for use.

The facility with which third mandibular molar teeth may be removed is based upon several factors. Density of the cortical bone (D) and the position

of the tooth (P) over the technique employed (T) will determine the ease of removal (R). $\frac{D + P}{T} = R$. Cortical bone protects the jaw; its destruction or impairment allows for the penetration of bacteria and other destructive agencies. If the tooth is a tissue impaction, its bone resistance is comparatively negligible; the position of the tooth, which must include the root formation, need offer little effort in the removal providing the technique is not at fault. Nothing should be taken for granted; the mobility of the tooth may be misleading. The incision should be made at the pterygomandibular raphe. Large flaps should be the rule to give an unobstructed view of the area of bone laid bare to permit its removal, when necessary, with care, and deliberation and accuracy, and to give visual and instrument access to the imprisoned tooth. The mucoperiosteum is held back from the bone by the blade of a Seldin elevator or by a Thoma tissue retractor, both serving its purpose well here. A straight line spear-pointed elevator used as a wedge will determine whether the case warrants full surgical procedure, for ofttimes the tooth may be elevated out of its socket with but a gentle half-round turn properly executed by this instrument. However, if resistance is offered, the flap operation should be the method of choice. Any proved technique suitable to the surgeon's skill may be employed. Some rely solely upon chisels, or the impactor, others on surgical burs (Shamberg or Hu Friedy), still others on both.

In passing, it might be in order to mention the splitting of the roots of impacted teeth when the x-ray reveals clearly defined roots. A knife-edge stone, tapering fissure burs, or impactor may be employed with caution to groove deeply the occlusal surface or the center of the buccal surface for at least ⅛ to ¼ inch, and with a twist of a bone chisel or Seldin elevator inserted in the prepared groove the tooth will be snapped in half, greatly facilitating removal. Others may strike off the mesial occlusal angle, allowing for an easy removal, and still others remove the entire crown of the tooth. These methods, it may be stated, eliminate much bone chiseling and resultant trauma. Whichever method is employed does not matter as much as the end result—the least trauma with minimum shock and fastest uneventful recovery.

Care must be exercised during the removal not to harm the lingual plate, and equal care should be exercised during the manipulation of elevators so that injury may not befall the mandibular nerve which is often superficially located. This might otherwise result in parathesia, which proves very annoying to the patient and embarrassing to the dental surgeon. The second molar should not be used as the fulcrum; utilization of the mesiobuccal surface of the process is safer, for it eliminates possible fracture of the distal root of the second molar tooth and its eventual loss.

Excessive numbers of surgical instruments employed can prove disconcerting and annoying. It would be well for knowledge of more than a few pets to be known, for every trick in the bag and every instrument may sometimes be needed toward the ultimate removal of impacted third mandibular molar teeth. It has been my habit to work from the buccal surface, for early experience had indelibly impressed the need for caution and for keeping clear of the lingual surface of the jaws. When dealing with an actively infected, impacted tooth it

is well to remember that the resultant trauma sometimes can force the pus and inflammatory products into the underlying tissues, breaking down any protective barriers set up by the leucocytes in the floor of the mouth and neck. A period of five days is considered safe to let nature build this protective wall of leucocytes. From here the infection may spread by the fascial planes to the sublingual and submaxillary regions and eventually to the pharyngomaxillary spaces, then downward to the mediastinum. Death may result either from suffocation due to edema of the larynx or from mediastinitis.

We must ever be mindful that hypodermic needles too are important in breaking down natural barriers, thus forcing infection into adjacent unaffected areas.

The third mandibular molar tooth completely embedded does not offer the same pathologic annoyances unless the patient has neurologic symptoms. It is those of the partially impacted variety that cause the greatest concern. Operative trauma must be minimized, for unduly traumatizing an infected area will almost always bring forth an exacerbation and metastasis. Therefore, it is advisable to operate only when the lowest incident of the pathologic condition is ascertained. It is imperative that incisions should be made to establish drainage when fluctuation, which indicates localization of the infection, is present. Wait until the acute condition has subsided to obtain the best results. Vincent's infection must be eliminated as a preliminary procedure. Scaling and polishing adjacent teeth and improving the general hygiene of the area is a good precautionary measure. Pyorrhetic areas are an added source of danger, being somewhat osteomylitic in nature; any excessive trauma may provoke an exacerbation from a chronic dormant type to the more virulent and acute forms, such as Ludwig's angina and septicemia, on to the pneumonias with fatal results.

SEDATION

The frequency of the occasion when the patient undergoes emotional distress is well known enough to the oral surgeon so that he ought to have a basic knowledge of the therapeutic value of sedation. He must be able to apply this knowledge as a remedial aid in form of premedication in his preoperative and postoperative surgery. All unusual stresses must be relieved and one of our best agencies to do this is by the many drugs at our disposal which have been found useful. These may be given one to one-half hour prior to the operation and will afford the patient relaxation as well as aid in recovery by thus minimizing shock. Hunger and exceedingly warm temperatures aid materially in causing the patient to become restive and an easy prey to psychic disturbances when a local anesthetic with adrenalin as one of its constituents is used.

The surgeon must make every effort to reduce the hazards to a minimum before the operation. He must reassure the patient and appreciate his frailties and shortcomings; in short, he must possess a bit of the milk of human kindness.

The matter of preoperative sedation, especially in the nervous and the apprehensive patient, depends somewhat on the choice of the anesthetic agent to be employed. The various barbiturates, notably luminal, sodium luminal, amytal, sodium amytal, nembutal, and so on, usually in rather large doses either by mouth or intravenously, are fairly widely used. We msut not lose sight of

the fact that they are toxic and habit forming. Their greatest benefit is probably in dulling the sensibility of the patient who is to undergo local or regional block. Barbiturates are not the perfect drug by far, but their value in oral surgery must not be denied.

While it is proper to prepare the patient by premedication, it is also important to relieve pain after the operation. Furthermore, the first duty of the surgeon is to prevent undue suffering. Morphine and codeine still remain the two absolutely dependable and reliable drugs. Administration must be carefully controlled, for they have habit-forming qualities and also act upon others with toxic effect and at times produce violent reaction.

Here again the barbiturates may be employed with good results. Eight grains of barbital or soluble barbital, 1½ grains of nembutal before operation and 3 grains before retiring; 3 grains of sodium amytal and 1½ grains of pentobarbital are the suggestive dosages for these drugs.

DIET

Diet must be assumed to play an important role in the recovery of ambulatory patients, particularly in maintaining their morale at a high level. Therefore the food intake should be of such quality, variety, and quantity to sustain a man of normal weight. It has been proved that 2,000 to 2,500 calories are sufficient for this. The nature of the diet should be of high caloric value, principally of proteins and fats. The carbohydrate intake is not of much importance at this time. It is important that the food be of the nonconstipating variety—dairy products, stewed fruits, cooked cereals, light soups and broths; when meat is used the fibers should be mashed or scraped to form an edible pulp. The food must be served hot at all times. Taxing the jaws by excessive chewing can only serve to delay healing and greatly inconvenience the patient. Along with a helpful diet, rest and plenty of sunshine also aid materially. Forced fluids are advisable.

POSTOPERATIVE TREATMENT AND HEALING

If it were possible to immobilize the jaws after a surgical removal, faster recovery could be expected. Suturing surgical flaps will also help. Healing in the area of third mandibular molars is not usually by primary intention, for the loss of the tooth is analogous to excessive loss of tissue. The best that can be hoped for is an uneventful healing by secondary intention. Before there can be any formation of healthy granulation tissue, there must be a total elimination of the infection present. This calls for débridement of all necrotic, diseased tissue and tissue severely traumatized during the surgery. At times there may be an outpouring of a purulent exudate brought about by the destruction of polymorphonuclear leucocytes together with tissue debris, "laudable pus," which may stimulate defensive mechanisms of the body, but at the same time recovery is retarded. This, however, is not always the case, for further penetration of the free pus into the deeper structures may bring on results other than what was bargained for. Septic hematomas are a detriment and must be removed before restorative processes are brought into play. Furthermore, unhealthy conditions of the mouth in general will increase the virulence of the bacteria; hence the elimination of such influences must be instigated.

After removal, we are confronted with the repair of the soft as well as the bony tissues. Were it at all possible to bring the healthy tissues into apposition the process would be simple and of short duration; there would be a gluing of the surfaces by coagulum containing the necessary ingredients for regeneration and reorganization. Sometimes the removal of the second as well as the third mandibular molar tooth will cause less postoperative trauma and allow for better approximation of the tissues. The clot is replaced by fibrous and osseous tissues. Sufficient periosteum and endosteum containing osteogenic properties will give a faster and a more favorable result than where the osteogenic tissues are of insufficient amounts. "Fibrils arising from the osteoblasts or bone cells send their prolongations in all directions and embed themselves into a homogenous matrix. Later deposition of calcium salts into this osseous tissue form the 'callus' which at first is spongy. Partial absorption and further deposits strengthen and restore the bone to its original contour."[4] The softer tissues on the surface form the scar tissue, which does not possess the tonicity and elasticity of the normal.

In the presence of much infection, depending upon the virulence of the invading bacteria, the weakened resistance of the host, or after a long protracted and difficult extraction, it is found expedient to use a wick in the socket. A 5 per cent iodoformed gauze saturated with a paste made from powdered aspirin and tinctures of merthiolate are used daily for the first three days, after which time the iodoformed gauze alone is used for another week and longer if necessary, changing the dressing every second or third day. Tight packings prevent formation of healthy granulation tissue and normal healing. They may also set up inflammatory disturbances detrimental to uneventful recovery, sometimes ending in sloughing.

In tissues where there has been little or no infection, gentle washing out of the socket with warm water to remove the food debris, followed by dusting or spraying with powdered sulfanilamide daily from five to eight days, will suffice. Pressure saline sprays are not necessary. Excessive pressure may sometimes prove harmful by forcing surface bacteria into the deeper structures. All the necessary bactericidal action will be found in the sulfanilamide powder.

Recent literature points to the replacing of sulfanilamide by sulfathiazole as the drug of choice for dental use. "It may be said that sulfathiazole is prepared for total use in all surgery because of its (a) greater solubility; (b) wider bacteriostatic effect on the organisms which may be encountered; (c) good phagocytic response; (d) less clumping into insoluble masses; (e) more prolonged effect."[15]

This author is of the opinion that sulfathiazole clumps readily into insoluble masses, more so than the other sulfonamides. Experience has shown that this drug will not be taken up readily by the tissue fluids, and therefore does not effectively exert its bacteriostatic effect topically and may even act in symbiosis with the infection present. Sulfanilamide, on the other hand, is quickly absorbed, which sometimes accounts for its toxicity, and therefore must be considered as the drug of choice when used topically as a spray for any surgery of the oral cavity.

It must be remembered that sulfathiazole must be given internally with

care, particularly to flight personnel. It may mean the grounding for as long as five days after the last dose had been administered. After long internal administration of the sulfonamide, perception is appreciably weakened and judgment warped; reflexes are sluggish and senses in general dulled. Periodic blood determination for sulfonamide content, when taken, will determine the duration of the grounding for the flight personnel.

Sulfadiazine, recently developed sulfonamide used to prevent infection and toxemia, has been brought into general use. A close check must be kept to prevent the development of its overconcentration in the blood stream, as absorption takes place rapidly. It is less toxic than any of the other sulfonamides. At first it was thought to be as good but no better than the other drugs, but more recent work indicates that it is doing a better job than first thought, and has resulted in many startling recoveries.

PREOPERATIVE PROPHYLAXIS

It has been the policy of the department here at NAAS, Chase Field, Texas, to treat prophylactically with sulfadiazine, along with the other therapeutic measures, for several days prior to removal, all cases in which there is acute pericoronitis of the tissues surrounding partially impacted third mandibular molar teeth. The elective dosage here is 15 grains of the sulfonamide, three times a day, along with 10 grains of bicarbonate of soda to compensate for the loss of the alkali reserve. The only untoward result since the inception of this activity was that of Cadet A, March 3, 1944, aged 22 years, who had failed to receive preoperative treatment. The tooth was removed as an emergency measure. Because of the unpleasant sequelae which developed postoperatively, it was suspected that the instruments employed might have been improperly sterilized.

History.—The removal was difficult, the trauma was doubtlessly considerable. The patient was hospitalized for twenty-four hours so that he might have the advantage of an ice pack, the use of sedatives, and proper postoperative care. He was discharged the next day and advised to return for further treatment.

Upon his return several days later he was definitely toxic, complained of considerable pain, slight chills, malaise, and had a temperature of 100.5° F. His white blood count was 10,750 (polymorphonuclear, 72 per cent; lymphocytes, 27 per cent; monocytes, 1 per cent). Swelling was extensive. Trismus and foul breath were present. The skin was drawn taut and did not pit under pressure. From the socket flowed a thin serous discharge. The patient was visibly ill and was immediately hospitalized.

Diagnosis.—Cellulitis.

Treatment.—Intravenous infusion of 500 c.c. of 5 per cent glucose in normal saline solution was instigated with warm moist magnesium sulfate packs applied externally. For a period of seventy-two hours warm irrigations of 0.7 per cent suspension of sulfathiazole in normal saline solution every twenty minutes was employed, and these irrigations were continued hourly thereafter until the patient was dismissed.

Chemotherapy.—Sixty grains of sulfadiazine along with 40 grains of bicarbonate of soda was used as the initial dosage, to be followed later four times daily by further administration of 15 grains sulfadiazine and 10 grains of bicarbonate of soda. The patient tolerated the drug favorably for a period of five days, after which time the use of sulfadiazine was discontinued. There was no means available to determine the blood level of the sulfonamides at the dispensary at that time. However, any manifestation of intolerance for this drug in the form of exfoliative dermatitis, bulbous skin, and mucous membrane involvement, granulocytopenia, or any other disturbance that might have been caused by the heavy con-

centration of the sulfonamides in the blood stream, was carefully watched for. And to combat this, large quantities of fluids were given which acted as a diuretic, thus activating a greater number of tubules in the kidneys so that the elimination of the sulfonamide crystals which might cause blockage of the renal tubules could easily be accomplished, and also to allow for the removal of toxic elements.

After the third day the swelling began to subside. As there was no fluctuation in any one or more areas suggesting the collection of pus beneath the surfaces was sufficient to warrant its release, no external incision for drainage purposes was deemed necessary. While none of the symptoms at the onset was unduly alarming, it was felt that the lowered resistance of the patient and toxicity present might encourage the disease to reach its critical stage and possibly develop serious consequences, had not the above measures been undertaken immediately. The patient was dismissed March 11, 1944.

Some authorities warn against the dangers of the use of sulfadiazine in the tropics. Patients under these environmental conditions lose large amounts of fluid through the skin, and several hospitals have reported patients with renal complications from the use of sulfadiazine due to low urinary outputs.[16]

Now very much in the limelight and given a great deal of publicity is the drug penicillin, which may prove superior to the sulfonamides for internal medication to prevent and minimize the ravages of infection of the head and neck. The results obtained both clinically and experimentally have been very gratifying and, with penicillin becoming available, it might conceivably be the chemotherapy of the future.

Since this paper was first undertaken many pertinent facts have been brought to light concerning the drug penicillin in its application toward combating debilitating sequelae of a dental nature. It had been found that it may be used safely empirically or in proved prophylactic dosages until the etiology of an infection may be fully determined. This drug has more than justified its reputation it now holds as the "wonder drug." Its cures have been many, rapid, and spontaneous. Penicillin has proved remarkably effective in the treatment of infections produced by the *Staphylococcus aureus,* hemolytic streptococcus, pneumococcus, and the gonococcus. It does have limitations, however; in general, the drug is not effective against gram-negative organisms; exceptions to this rule are the gonococcus and meningococcus. Craig et al.* have demonstrated it as the drug of choice and have described the present status of penicillin, its various methods of employment, and suggested dosages in the treatment applicable to infections in general, and particularly to those infections arising in the head and neck of dental origin. Dosages of 15,000 to 30,000 units are administered every two or three hours intramuscularly, from four to seven days in acute infections, and from seven to twenty-one days in subacute and chronic infections. This need not be a hard fast rule as the amounts employed are determined by the needs of the patient.

Purulent material must be evacuated to get full beneficial results. Penicillin also works to advantage in symbiosis with the sulfonamides. Using it topically, as much as 100,000 to 10 grams may be used at one time. It is not nor can it be used as a substitute for careful surgery. It does, however, allow for a greater margin of safety in all surgical procedures when employed preoperatively and postoperatively. The drug must be given around the clock to maintain a continuous penicillin blood level, so that it may inhibit the individual affecting organisms until the end of the treatment.

If facilities are available, a small amount of the infectious material can be cultured and will determine the amount of the drug to be used. A diet of high caloric and protein content is necessary to maintain the nitrogen balance.

If after twenty-four or forty-eight hours penicillin has not given relief in cellulitis, then the purulent areas must be sought out and proper drainage instigated. Total penicillin dosage may vary from 290,000 units in cellulitis of the face following dental extraction, to 2,141,050 units for a tense phlegmon, Ludwig's angina, not infrequently of dental origin.

*Craig, Capt. Winchell M., et al.: Navy Medical Bulletin 44: 468, No. 3, March, 1945.

The toxicity of penicillin is remarkably low. Because the material available for clinical trial is not a pure product and is of a varying composition, it seems feasible that such reactions as have been observed clinically may have been due to impurities rather than penicillin itself. Urticaria also has been observed in a few instances, but in most cases the responsibility of penicillin for the eruption has not been clearly established.

In the recent findings of Herrel and Nichols[17] of the Mayo Clinic, who had administered penicillin to six patients with cellulitis of the mouth, two of whom had a definite bacteremia, the resolution of the local inflammation and the improvement in the toxemic state was prompt and permanent.

Preoperatively supplementing the above treatment, ice packs are used externally over the area, which causes constriction of the peripheral vessels, thus tending to prevent absorption of the toxic elements. They may also abort swelling and inflammatory processes and by so doing produce an analgesic effect. Ice packs are used very effectively postoperatively as well. If pain persists, stimulation by mild heat in the form of infrared rays will help materially, causing a marked degree of hyperemia to the depth of the penetration of the light ray. Interchangeable application of heat and cold is sometimes productive of much relief to the patient.

Even after healing processes have terminated and pain still persists, especially during mastication, all available means at the oral surgeon's command failing, diathermy might be the means of choice to furnish the necessary relief. The mode of application allows for deep penetration of heat and can reach the source of trouble when all other avenues are closed.

"Medical diathermy creates an intense internal heat, localized at will, with a subsequent active hyperemia which greatly reduces repair time and aids in the resistance to localized infection. Its general effect in decreasing pain and promoting general and local metabolism is also made use of."[19]

CONCLUSION

In conclusion, the clinical findings in 1,031 case histories carefully studied have brought out some pertinent facts. Sixty-five per cent of all third mandibular molar teeth can be utilized. However, the exigencies of war and circumstances do not allow for conservative treatment in many cases. It can readily be seen that, as a result, many of these teeth will be lost, which under normal circumstances might have been saved.

Removal of this tooth is indicated in the following cases: (1) existence of pericoronitis; (2) insufficient space for the tooth to erupt normally; (3) when this tooth interferes with the future successful use of dental prosthetic appliances; (4) neurologic and cystic involvement; (5) considered foci of infection; (6) when the tooth does not respond to conservative therapeutic measures; (7) when a satisfactory filling cannot be placed in the tooth due to extensive caries; (8) pyorrhea; (9) intermittent history of pain when bodily resistance is at a low ebb and the tissues surrounding are in a continual chronic inflammatory state; exertion of excessive pressure by this tooth on the teeth adjacent sufficient to cause abrasion of the interproximal surfaces and also interference

with the proper alignment of the other teeth. This excessive mesial pressure may be sufficient to cause extensive decay of the distal surface of the second mandibular molar teeth.

It would be wise for the dental surgeon to steal a leaf from the book of the medical profession. If the x-ray findings reveal an impacted tooth that appears difficult to remove, it is advisable to dismiss the patient and carefully study the findings. This may save much exertion and anxiety. No attempts to read the wet negative should be made; instead, the negative should be carefully dried and placed under the proper illumination of the magnifying light and viewing box and studied. The viewing box will reveal much that would ordinarily be overlooked. Never attempt a difficult removal at the end of a trying day. A good night's sleep will sometimes mean the difference between success and failure.

In concluding this paper, it is interesting to note that Wald, of the U. S. Naval Hospital, Brooklyn, New York, states that in 100 cases of cellulitis of the head and neck of dental origin, 95 of these were definitely attributed to the removal of the third mandibular molar teeth, the remaining 5 per cent to other teeth and causes.[19]

It is desired to express gratitude to William O. Huie, Yeoman First Class, U. S. Naval Reserve, for his splendid cooperation in the preparation of this paper, to Lieutenant C. A. Potts, U. S. Naval Reserve, for the excellent photographs of the graphs, and to Lieutenant L. Sagharian, Dental Corps, U. S. Naval Reserve, for his constructive criticism and helpful suggestions.

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EXTREME LATERAL EXCURSION OF THE MANDIBLE SIMULATING DISLOCATION

Case Report

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New Haven, Conn.

J. V., a 13-year-old girl, was seen in the Dental Clinic of the New Haven Hospital with a complaint of pain from the mandibular right first molar. A diagnosis of acute pulpitis due to a pathologic exposure was made by the examiner. The diseased tooth was extracted under local mandibular block anesthesia.

Five days later the patient returned with a complaint of increasing pain in the right mandible. The alveolus was irrigated with a warm sodium perborate solution at this time.

Nine days later the patient returned with the complaint of moderate pain, tenderness, and swelling of the right cheek. The patient stated that she had been unable to use her jaw properly to eat solid foods for the past ten days. The objective signs were: deviation of mandible to the right side, trismus, temperature of 101.6° F. There was considerable extraoral swelling in the right cheek. Intraoral inspection showed a swelling in the right retromolar region. In closed position, the mandible was in right lateral excursion to the extent of the width of the mandibular central incisor and one-half of the mandibular lateral incisor (Fig. 1).



Fig. 1.—Photograph showing extreme lateral excursion of the mandible to the right. Note maxillary midline bisecting incisal portion of the left mandibular lateral incisor.

The following clinical diagnoses had been recorded:

1. Infection of the right buccal tissues with a dislocation of the mandible to the right.
2. Involvement of masseter and pterygoid muscles.
3. Trismus.

The patient was hospitalized. Hot packs and soaks were applied extraorally and hot irrigations intraorally. Lateral jaw and anteroposterior head roentgenograms were taken and sedation with codeine or morphine p.r.n. was ordered.

The radiographic report read as follows:

"Roentgenographic examination of the right first molar area and the mandible reveals a double-grooved area of rarefaction as the result of the recent extraction of the first molar. No definite retained root fragments are noted, nor is there any evidence of osteomyelitic involvement at this site. It is noted that the third molar is unerupted and that the crown of this tooth is impacted against the neck of the second molar. Although there does not appear to be any displacement of either condyle from its respective mandibular fossa, the P. A. projection shows a deviation of the mandible to the right so that there is a definite space between the teeth on the right side, with an overlapping of the teeth in the

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analogous position on the left. This thrust of the mandible, caused presumably, by the soft tissue swelling on the right, while it has resulted in no actual dislocation of the condyles, has caused a forward displacement of the left condyle within its fossa and a posterior displacement of the right condyle, so that the normal joint space is partially obliterated in both instances. There is a slight haziness of the outlines of the right temporomandibular joint suggesting the possibility of an early infectious arthritis of the same. It is possible, however, that this haziness is due to the thickness of the adjacent swollen soft tissue.

"Radiographic impression: The findings are negative for evidence of osteomyelitic involvement of the mandible. The deviation of the same to the right is not of a sufficient degree to produce a dislocation of either condyle from its harboring fossa." (Fig. 2.)

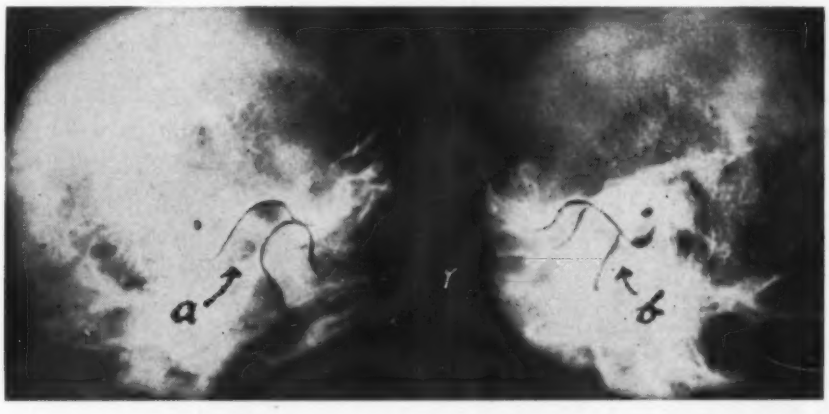


Fig. 2.—Roentgenogram showing: (*a*) head of left mandibular condyle in extreme forward excursion; (*b*) right condyle apparently resting directly on the articular surface of the glenoid fossa with obliteration of the normal space commonly seen between fossa and condyle.

The swelling of the face increased for a period of two days after admission but decreased following the spontaneous rupture of an abscess into the oral cavity with a copious discharge of pus. Examination now revealed that the abscess was located on the medial surface of the right ascending ramus and had ruptured at the site of the mandibular injection. The patient experienced considerable relief from pain and further sedation was now unnecessary. The trismus and deviation of the mandible to the right, although still present, showed a daily progressive improvement. At the time of discharge from the hospital, the pain and swelling subsided, the patient could open her mouth comfortably about 3 cm., and could bring her teeth into centric occlusal relationship at rest.

It seems possible that the infection was introduced in this patient at the time of the injection of anesthetic solution, when pathogenic bacteria may have been transferred from the mouth to the underlying tissues. The hard swelling with abscess formation caused pressure between the posterior portion of the right maxilla and the external pterygoid plate of the sphenoid bone medial to the mass, and the anterior portion of the internal aspect of the right ascending ramus of the mandible lateral to it. This mass may have served as a fulcrum and together with the right pterygoid muscles, which may have been in spastic contraction, caused a rotation of the body of the mandible toward the right.

This case closely paralleled that of another patient seen in the clinic. There was a similar condition of trismus, with displacement of the mandible to the right the width of the lower central incisor from the maxillary midline, with the presence of soft tissue infection in the region of the anterior portion of the ascending ramus. However, there was no spontaneous rupture into the oral cavity but, rather, a fluctuant area located over the right masseter muscle which eventually required external incision and drainage. The operation was followed by healing and a return of the displaced mandible to centric occlusion.

These cases suggest the possibility that a displacement of the mandible within its normal range, simulating unilateral dislocation, may be brought about by infection introduced in the course of a mandibular block injection.

ORAL MANIFESTATION IN VON RECKLINGHAUSEN'S DISEASE (NEUROFIBROMATOSIS)

With a Case Report

Max H. Jacobs, M.D., D.M.D., Boston, Mass.

FIBROMAS of the oral mucous membranes are common. Neurofibromas in relation to the mandibular or mental nerves, though not common, have been described and reported. An occasional solitary neurofibroma has presented itself for description. The neurofibroma of the oral mucous membrane as part of a generalized neurofibromatosis, although not exactly rare, has seldom been described, as a result of which, this case is being reported.

In 1882, Von Recklinghausen described a condition characterized by multiple tumors of the skin, associated with anatomic abnormalities in the central nervous system, vascular system, bones, and endocrine glands.

The etiology of this disease is obscure. Trauma as a causative factor has been suggested. The numerous cases reported in the literature fail to point to trauma as evidence in connection with the development of the condition.

There is evidence to support the contention that neurofibromatosis is familial and hereditary. Ewing[1] quotes Preiser and Davenport as having fully investigated the hereditary factor and found "that it behaves as a dominant, equally affecting both sexes and transmitted by both." Ewing[1] also quotes Harbitz as having traced the disease through five generations.

Stelwagon,[2] having studied the observations of Virchow, Konigsdorf, Oeterlony, Atkinson, and others, who reported the disease to have occurred in several successive generations, or sometimes in more than one member of the same family, concluded that heredity or family tendency must be considered a factor.

There has been some discussion as to the point of origin of the neurofibroma. Bell[3] feels that the abundant formation of collagenous fibers suggests that the tumors originate from the connective tissue of the nerve trunk. He states that the diagnosis of neurofibroma is established by the microscopic demonstration that a nerve penetrates the tumor.

Masson[4] concluded that the origin of the neurofibroma is from the cells of the sheath of Schwann and calls them Schwannomas. Most of the evidence presented in the literature points to the sheath of Schwann being the source of origin.

The disease manifests itself as early in life as infancy. It appears to occur in phases, the form it takes depending upon the organs and structures involved.

For many years, the patient may present flattened, slightly raised, brownish-yellow pigmented areas scattered over the trunk and extremities. These pigmented areas have been termed the *café au lait* spots of Von Recklinghausen.





Figs. 1 and 2.—Von Recklinghausen's disease.

This points to the probability of some endocrine disturbance, such as formation of a tumor near or within the adrenal gland. In some cases, no other manifestation of the disease except these pigmented areas may take place. In others, bone disturbances, such as osteomalacia or acromegaly, may occur. This again points to probable endocrine disturbance, such as tumor formation within or near the pituitary gland.

The pigmented patches precede tumor formation. Yet patients may present multiple neurofibromas diffusely over the body without any history of previous manifestations of pigmented areas.

In the brain, the disease is associated with gliosis, glioma, or sclerosis. When the spinal cord is involved, spina bifida or meningocele may be present. The mucous membranes are not immune and the neurofibromas have occurred on the lips, cheeks, palate, and jaws.

The tumors vary in size from pinhead to massive proportions, generally being seen on the skin as pea to grape size. Larger tumors, especially those exposed to irritation, may undergo sarcomatous degeneration.

In the jaws, the neurofibroma originates from one of the peripheral nerves and may increase in size unnoticed, unless periodic dental examination takes place.

The following case illustrates the necessity for frequent dental examination and the thorough microscopic examination of all tissues removed from the mouth and jaws.

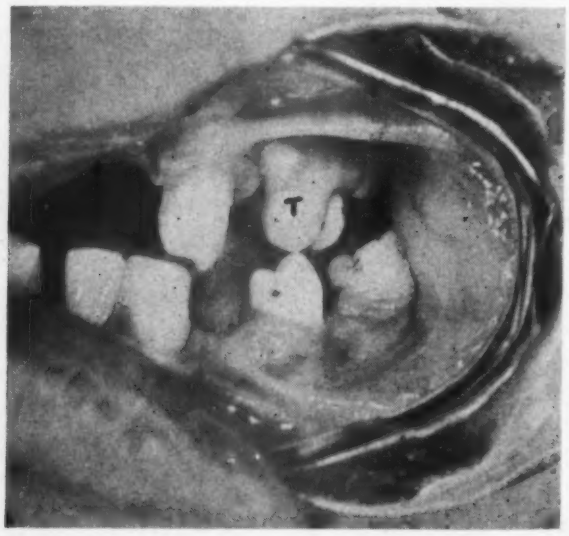


Fig. 3.—Tumor *T* attached to buccal gingiva partly covering tooth.

CASE REPORT

R. R., white woman, aged 43 years, presented herself because of a lump in the roof of her mouth.

Twenty-five years before she had first noted small growths covering her arms, legs, body, and face. In the last ten years they had become larger and more numerous. There had been no pain or bleeding associated with these growths. Four weeks before she first noted a lump in the roof of the mouth which bothered her when eating or swallowing.

Examination disclosed a rather poorly nourished, well-developed woman, whose trunk, legs, and face (Figs. 1 and 2) were covered by hundreds of tumorous growths ranging in size from pinhead to grape. Some of them were hard, while others were soft. Manipulation of the tumors caused no pain or bleeding.

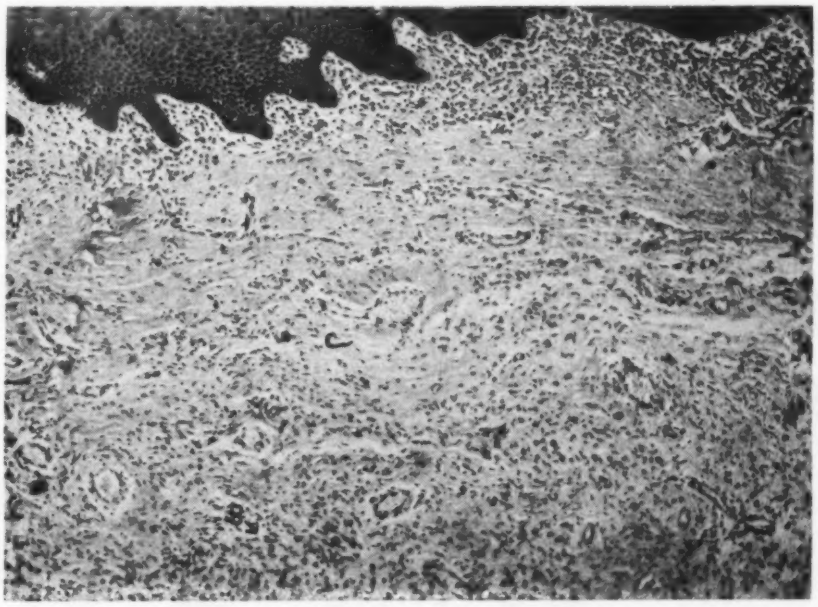


Fig. 4.—*A*, Stratified squamous epithelium. *B*, Palisade units. *C*, Connective tissue stroma demarcation between oral mucosa and tumor.

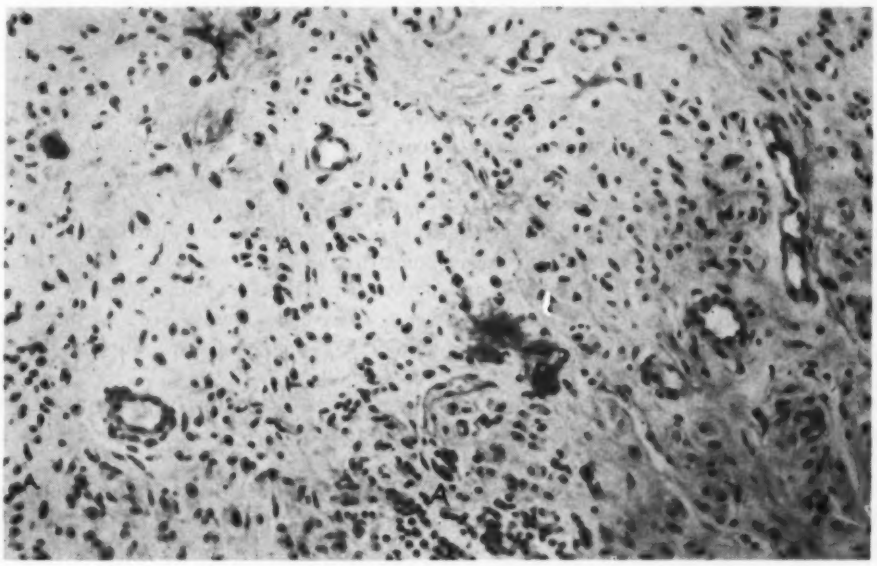


Fig. 5.—*A*, Palisade units within tumor.

At the junction of the hard and soft palates, in the midline, was a lesion about 1 cm. in diameter and raised 1 cm. above the surface of the mucous membrane. It was soft but nonfluctuant, had a distinct circumscribed border, and was superficially ulcerated.

In the left maxilla was a hard cone-shaped mass extending from the mesial surface of the first molar to about the cuspid area, and extending downward in the shape of a nipple,

so that on mastication the apex of the tumor reached the occlusal surfaces of the mandibular teeth (Fig. 3, *T*). (She had not known that this tumor was present.)

X-ray examination of the palate and jaw disclosed nothing of note.

The mass in the palate was excised first. The microscopic examination was consistent with that of chronic inflammation.

The maxillary tumor was clinically diagnosed as fibroma, possibly neurofibroma. Because of the pecularity of its shape, and the presence of multiple neurofibromas over the rest of the body, the mass was removed with wide margins, including the involved teeth and underlying bone.

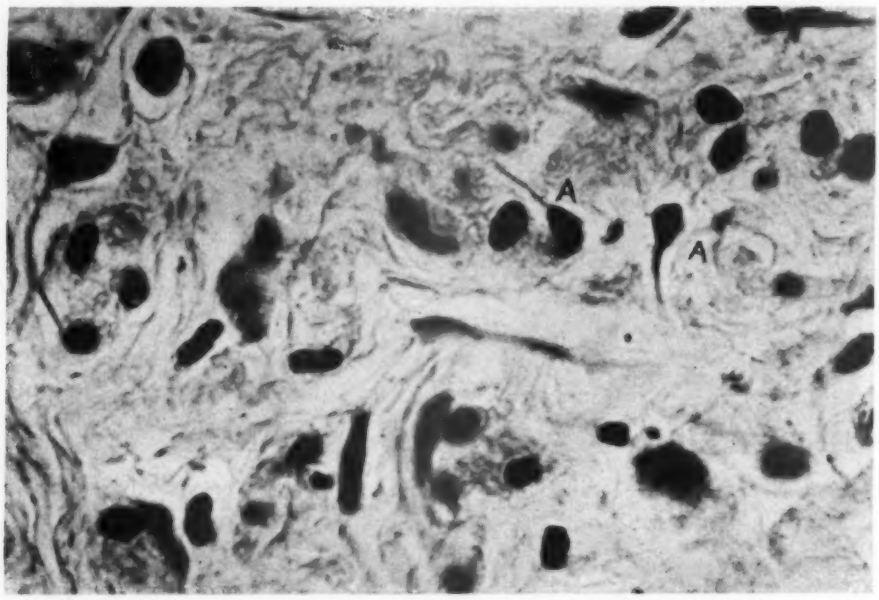


Fig. 6.—*A*, Schwann cells.

Pathologic Examination.—The specimen consists of a tumor mass to which underlying bone is attached. The tumor measures 1.5 cm. in length and 1 cm. in height. It is covered by normal-appearing oral mucous membrane.

The section is covered on one edge by typical stratified squamous epithelium which sends well-marked rete pegs into the connective tissue beneath it. This connective tissue, in places, is heavily infiltrated with small round cells and somewhat dilated capillaries.

Below this submucous connective tissue the tumor commences and is seen as more cellular elements. There appears to be a somewhat acellular layer of collagen fibers between the connective tissue and tumor tissue (Fig. 4).

The tumor mass consists of connective tissue stroma and contains large and small round cells. Scattered throughout are fibroblasts and palisade units. Some hyalinization occurs around the vessels (Fig. 5).

A striking feature within the tumor tissue is the presence of moderate numbers of deeply staining ovate nuclei, within pale staining cytoplasm, from which are extensions of fibrils (Schwann cells) (Fig. 6).

Diagnosis.—Neurofibroma.

SUMMARY

A case of neurofibroma of the maxilla as part of Von Recklinghausen's disease is reported. The literature is briefly reviewed and the histopathology discussed. The necessity for microscopic study of all tissues excised from the oral cavity is pointed out.

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311 COMMONWEALTH AVENUE

SURGERY OF THE MANDIBLE: THE AMELOBLASTOMA

LOUIS T. BYARS, M.D., F.A.C.S., ST. LOUIS, MO., AND
BERNARD G. SARNAT, M.D., D.D.S., CHICAGO, ILL.

THE ameloblastoma develops from the cells of the enamel organ during a particular phase of tooth development. It is found most frequently in the mandible, less so in the maxilla, and occasionally in the pituitary gland, tibia, ulna, and ovary. For the purposes of this report only the ameloblastoma of the mandible will be considered. Although this tumor is considered to be uncommon there have been over 400 reported.[1, 4-6] Both sexes are affected in about equal frequency. The ameloblastoma has been recognized at birth and as late as the seventy-sixth year; the average age in Robinson's series was 37.6 years.[6]

The ameloblastoma is generally described as a benign neoplasm although, in a study of 379 tumors, 4.5 per cent showed metastases or histologic evidence of malignancy.[6] A better known characteristic of this tumor is its persistence and tendency to "recur" after operative intervention. The tendency for the ameloblastoma to "recur" locally is probably due to inadequate surgical treatment. The purpose of this report is to consider (1) the surgical treatment of ameloblastoma of the mandible and (2) the reconstruction necessary subsequently.

BRIEF SUMMARY OF DENTAL HISTOGENESIS

A brief review of dental histogenesis will facilitate understanding the origin not only of the ameloblastoma but also of other dental anomalies (Table I).

TABLE I. STAGES OF TOOTH DEVELOPMENT DURING WHICH CERTAIN DENTAL ANOMALIES FIRST APPEAR

STAGES OF TOOTH DEVELOPMENT	DENTAL ABERRATION
I. Growth	
a. Initiation	Anodontia
b. Proliferation	Odontome
c. Differentiation	
Histodifferentiation	Preameloblastoma (Ameloblastoma)
Morphodifferentiation	Hutchinson incisor, Moon molar
d. Apposition	Chronologic enamel aplasia (Enamel hypoplasia)
II. Calcification	Fluorosis
III. Eruption	Pseudoanodontia

Tooth development may be divided into three stages: (1) growth, (2) calcification, and (3) eruption. For the purpose of this report only the first stage (growth) will be considered. This stage can be further subdivided into (a) initiation, (b) proliferation, (c) differentiation, and (d) apposition (Fig. 1).

From the Department of Surgery, Washington University Medical School.

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Initiation.—Between the sixth and seventh weeks of intrauterine life an inward proliferation of the oral epithelium (*O.ep.*) begins (Fig. 1, *A*). This is the dental anlage (*D.a.*). If initiation does not occur there will be no tooth development and anodontia will be the clinical condition.[2]

Proliferation.—Cellular proliferation continues until an invagination takes place and the connective tissue which borders the invaginated epithelial portion condenses and forms the dental papilla (*D.p.*), the primordium of the dentine and pulp (Fig. 1, *B*). During this period the early form of the odontome arises.

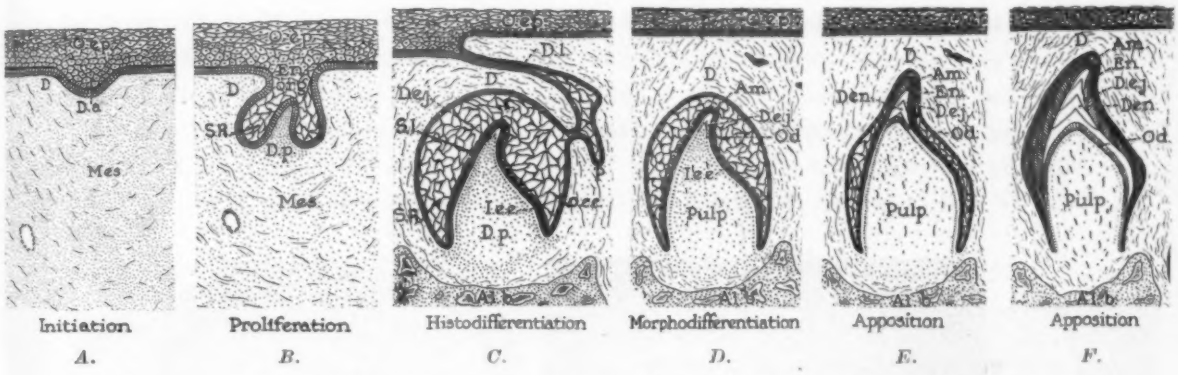


Fig. 1.—Diagrammatic representation of the embryology of the lower human deciduous central incisor (modified from Sarnat and Shaw[9]). *O. ep.*, oral epithelium; *D. a.*, dental anlage; *D.p.*, dental papilla; *En.org.*, enamel organ; *o.e.e.*, outer enamel epithelium; *S.R.*, stellate reticulum; *S.I.*; stratum intermedium; *I.e.e.*, inner enamel epithelium; *D*, deciduous incisors; *P*, permanent tooth bud; *Mes.*, mesenchyme; *D.e.j.*, dentinoenamel junction; *D.l.*, dental lamina; *Al.b.*, alveolar bone; *Am.*, ameloblasts; *Od.*, Odontoblasts; *En.*, enamel; *Den.*, den ine.

Differentiation.—The enamel organ (*En. org.*) which originates from the invaginated epithelium gives rise to the outer enamel epithelium (*O.e.e.*), the stellate reticulum (*S.R.*), the stratum intermedium (*S.I.*), and the inner enamel epithelium (*I.e.e.*) (Fig. 1, *C*). It is during this period that the ameloblastoma arises from the enamel organ. This occurs within the first few years of life although no activity may become manifest until some years later. Enamel is never found in this tumor. If tooth development continues normally, however, the inner enamel epithelium will differentiate into ameloblasts, the enamel-forming cells. Thus one can readily see that ameloblastoma is not the correct term because the tumor arises from the enamel organ before the ameloblasts have differentiated. The term preameloblastoma is more accurate.

In addition, the odontoblasts differentiate from the adjacent connective tissue of the dental papilla. The dentinoenamel junction which forms the basic pattern of the tooth is determined between the inner enamel epithelium and the adjacent connective tissue of the dental papilla (Fig. 1, *D*). It is during this period that the Hutchinson incisor and mulberry molar develop.[9]

Apposition of Dentine and Enamel.—In this stage a cusp of dentine is formed and with this stimulus the preameloblasts change to functioning ameloblasts and a corresponding amount of enamel is formed on the outer portion of the dentinoenamel junction (Fig. 1, *E*). When this formation has been initiated, there is a synchronous recession of the odontoblasts and ameloblasts from

each other. Incremental layers of enamel are apposed one on top of the other until the cusp is fully formed. Subsequent layers are apposed at the sides until the crown is complete (Fig. 1, *F*). Systemic disease during this period sometimes affects enamel formation.[7, 8] After formation of enamel has ceased, formation of dentine continues to complete the root.

DIAGNOSIS

Clinical Findings.—The ameloblastoma which is most frequently seen in young adults, is characterized by a slow progressive swelling of the jaw, usually near the angle. The tumor sometimes attains large proportions (Figs. 2, *A*, 3, *A*, 4), extends toward the clavicle and weighs several pounds. The growth may be accompanied by pain. There is usually a history of a tooth or teeth having been extracted from the area several years previously and of several attempts surgically to eliminate the tumor. Fistulous tracts leading to the oral cavity and secondary infection of the tumor are not uncommon.



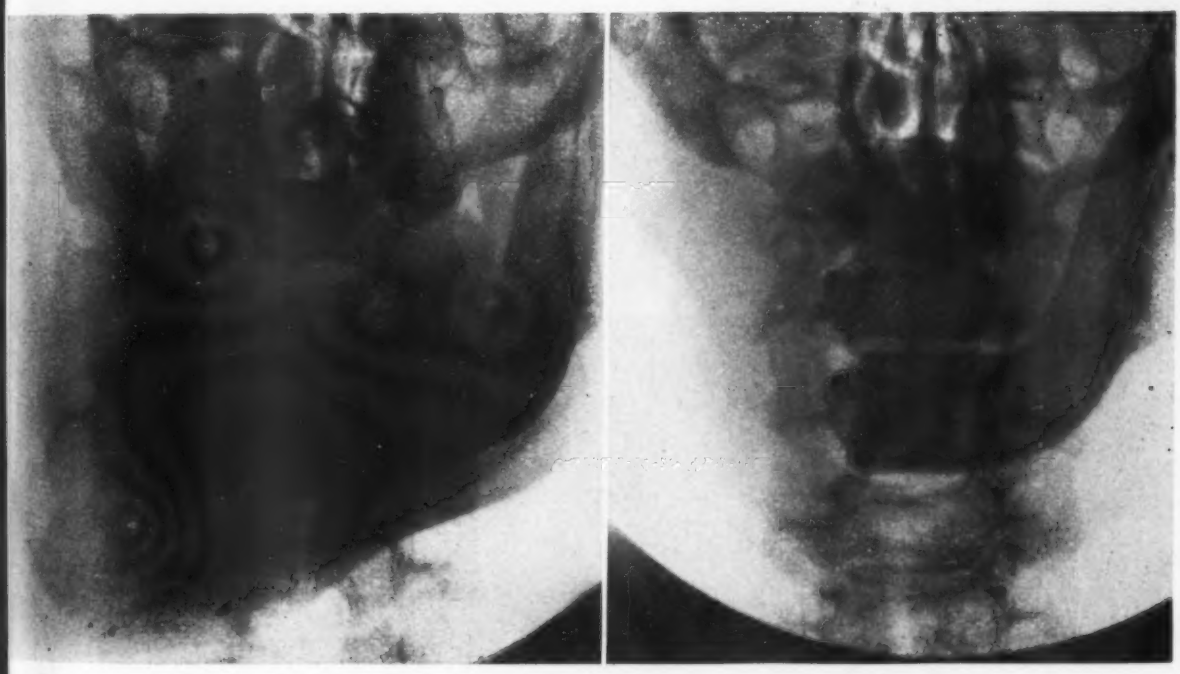
A. *B.*

Fig. 2.—*A*, Photograph taken on May 10, 1943, of a patient with a large ameloblastoma of the right side of the mandible. F. R., white male, aged 43 years, was first seen by us in May, 1943. Swelling was first noticed inside right side of mouth in 1915. The mass continued to grow and patient had at least three local intraoral operations. In 1938 the right side of the jaw suddenly began to swell and at that time the mass was about 6 cm. in diameter. The mass has continued to grow. In 1939 a diagnosis of ameloblastoma was finally made. Patient was told at that time that the operation would be worse than the disease and was told to go home and forget about it. When the patient was seen in 1943 he had been unable to work for the past few years. A hemisection of the mandible was done on May 11, 1943. He has worked ever since his recovery from the operation and has enjoyed good health. *B*, Postoperative photograph taken on May 25, 1943, after resection of the mandible.

The buccal plate of bone is usually expanded most, although the lingual bone may be expanded to push the tongue to the opposite side of the mouth. The bone is sometimes so thinned that it will crack like an eggshell. These are late findings, after the tumor has changed from the solid to the cystic phase. The pa-

tient may be seen only after pathologic fracture has occurred. Only occasionally do true signs of malignancy appear.

Roentgenographic Findings.—The roentgenogram serves as a valuable adjunct in the diagnosis of cystic lesions of the jaw, but the final diagnosis depends on the gross and particularly the microscopic examinations. The roentgenogram is most valuable for the early diagnosis of ameloblastomas of the jaw when the tumor is still centrally located, has not expanded the bone, and is in the solid phase. It is at this time, when most of the mandible is as yet not destroyed, that the tumor can be removed completely with no serious deformity resulting to the patient. When the ameloblastoma has become cystic and considerably larger, the clinical findings are obvious and the roentgenogram is of primary value in showing the extent of bone destruction (Figs. 3, *A* and 4, *A*). There is no constant characteristic roentgenographic description of the ameloblastoma (Fig. 5). The multilocular appearance and the scalloped border are by no means pathognomonic. The ameloblastoma, giant-cell tumor, carcinoma, and other lesions which are destructive cannot be positively differentiated from each other on the roentgenogram.[3]



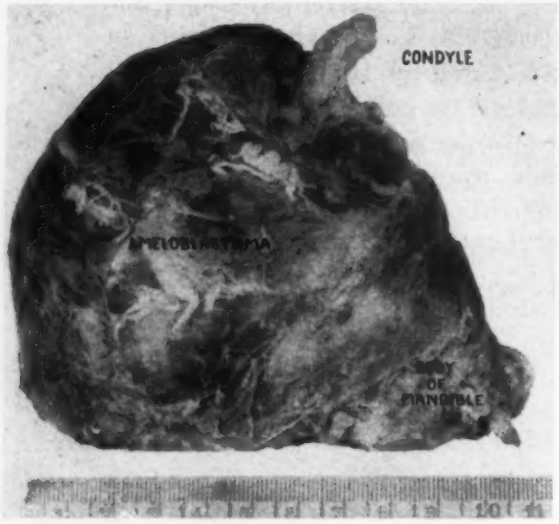
A. *B.*

Fig. 3.—Roentgenograms of same patient as in Fig. 2. *A*, Preoperative. Note the large mass on the right side of jaw and face. *B*, Postoperative, after resection of right half of mandible.

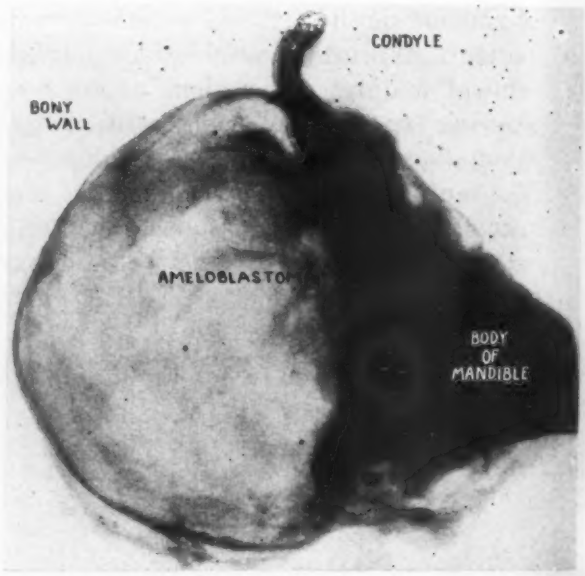
Gross Findings.—The findings on gross examination vary with the stage of development of the ameloblastoma. Analysis of 219 cases revealed that 57.5 per cent were cystic, 24.2 per cent were both cystic and solid, and 19.1 per cent were solid.[6] This classification is arbitrary and combinations of the solid and cystic tumors are found in varying degrees. The solid tumor which usually represents an earlier stage in development is of fine granular consistency, and

encapsulated. There may be one principal mass with numerous smaller daughter areas. The cystic type usually represents the solid tumor after cystic degeneration (Fig. 4, *C*). The contained fluid may be clear yellow to red and either of a mucous or serous consistency. The bone is expanded and may be parchment thin (Fig. 4, *B*).

Microscopic Findings.—Interpretation of the microscopic findings in the ameloblastoma is facilitated by an understanding of the histology of the enamel



A.



B.

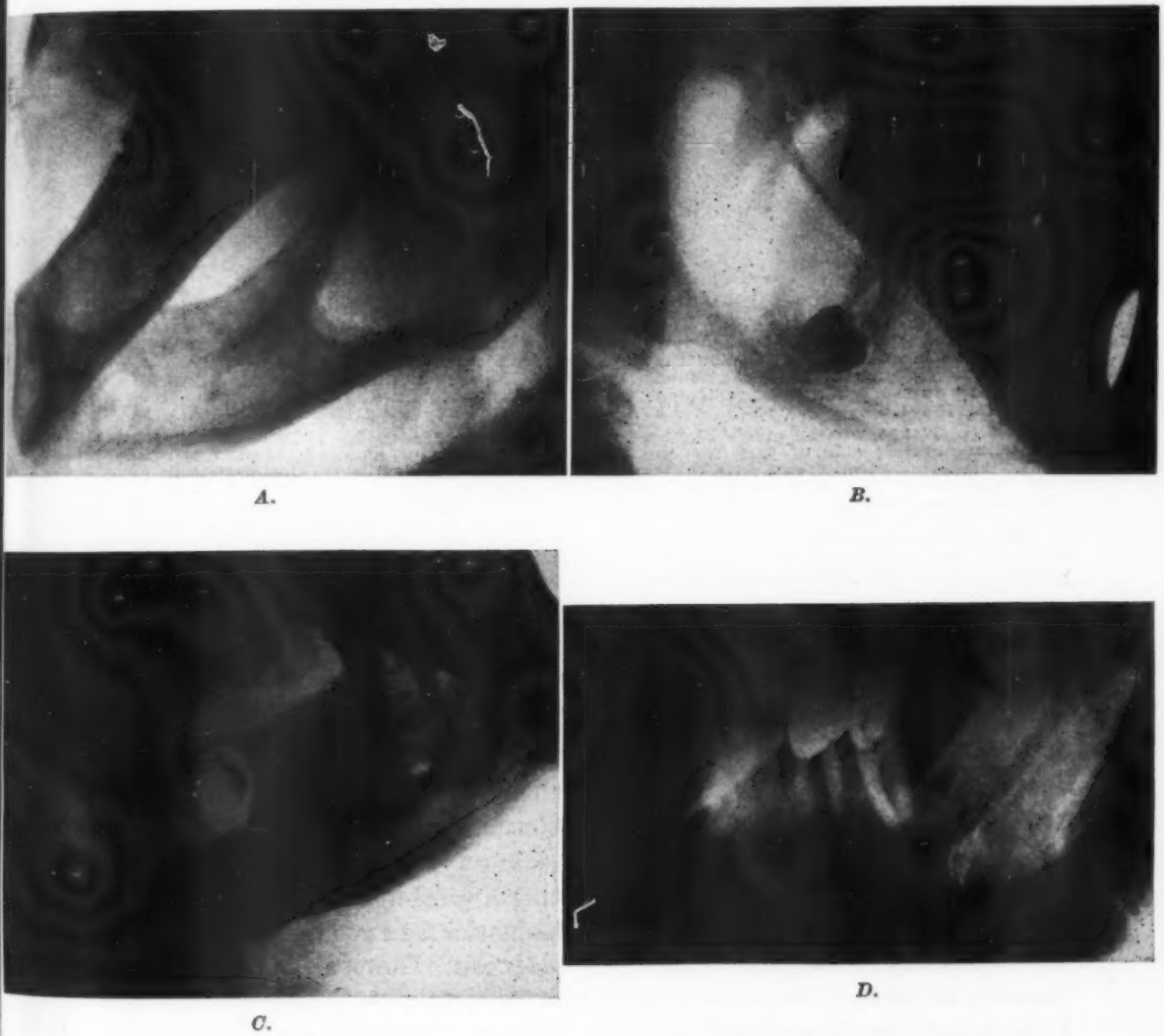


C.

Fig. 4.—Photograph, *A*, and roentgenogram, *B*, of the tumor and mandible which were resected. In *B* note the distortion of the condyle, ramus, and angle of mandible and absence of coronoid process. *C*, Photograph of specimen after it was cut in two. Note the large single cyst, the smaller cysts and solid tumor.

TABLE II. HISTOLOGIC CLASSIFICATION OF THE AMELOBLASTOMA[12]

TYPE OF AMELOBLASTOMA	CHIEF CHARACTERISTICS
1. Epithelioma	Little differentiation of epithelium which grows in cords or strands
2. Stellate	Epithelial cells stellate in shape; grow in sheaths or contained in follicles
3. Ameloblastic	Cylindrical cells (preameloblast) lining follicles
4. Acanthoma	Stellate reticulum differentiates into squamous or prickle cells, epithelial pearl formation
5. Carcinoma	Squamous cell



A. *B.* *C.* *D.*

Fig. 5.—Lateral roentgenograms of the mandible each showing different characteristics of the ameloblastoma. Although none of these are similar, each was proved to be an ameloblastoma on microscopic examination (*D*, courtesy, The C. V. Mosby Co.).

organ (Fig. 1). Thoma states there are many variations due to the stage of differentiation the epithelium may attain when the tumor formation began, the behavior of the stroma, and the malignant changes which may develop (Table II). Malignant degeneration, although uncommon, does occur (Fig. 6).

Simmons states that where the cuboidal cells predominate, the growth is more malignant than in the cystic type with well-developed ameloblast-like cells. Schweitzer and Barnfield believe that there is no apparent correlation between the histologic pattern of the ameloblastoma and its ability to metastasize.

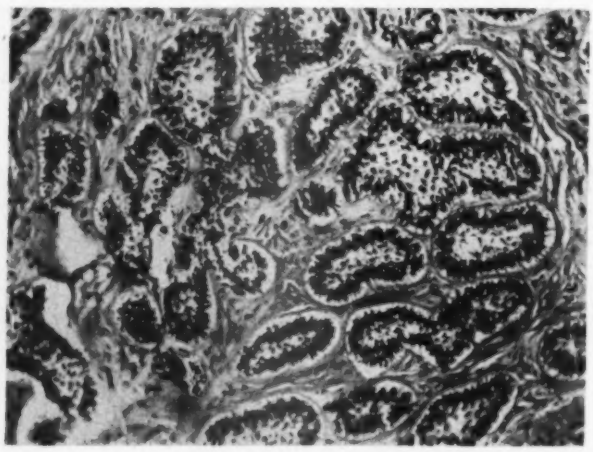


Fig. 6.—Photomicrograph of a malignant ameloblastoma (of the jaw) which eventually metastasized to the lungs. Note the characteristic arrangement of the stellate cells with poorly defined cytoplasmic boundaries. These are surrounded by columnar cells which are hyperchromatic and do not possess the regular arrangement usually encountered in the more slowly growing type of ameloblastoma. The epithelium is arranged in irregular groups surrounded by dense collagenous stroma which tends to form a pseudobasement membrane (Sept. 16, 1940).

O. N., Negro woman, aged 23 years, was first seen on May 23, 1928, with the complaint of swelling of left side of face for one year. A diagnosis of ameloblastoma was made and it was removed through an intraoral approach without resection of the mandible. Patient next returned in 1935 with recurrent swelling of mandible and was reoperated upon. Radium was subsequently implanted (2232 mg. hours). Nodules soon appeared along ear, orbit, and forehead and eventually involved the entire left side of head. Patient began to vomit blood in 1939 and examination of the chest plate revealed multiple nodules in both lung fields. Necropsy was performed in 1942 and histologic examination of the tumor in the lungs revealed it to be morphologically identical with the tumors in operative specimens.[10]

SURGICAL TREATMENT

Inasmuch as radiation does not affect the ameloblastoma materially, the treatment of this tumor of the jaw is primarily surgical. Both the surgical approach and the procedure which is carried out depend not only upon the diagnosis of the type of tumor but also upon its extent. In some instances the extent of the tumor will be such as to determine the type of surgical approach. In other cases, it is first necessary to know the type of tumor with which one is dealing and to determine, subsequently, the surgical approach and procedure on the basis of this knowledge. Clinical examination and roentgenographic studies will sometimes be adequate to make the diagnosis. However, in a large group of mandibular tumors diagnosis will still not be certain even after examining both patient and roentgenograms.[3]

In this latter group either preliminary biopsy or examination of a frozen section at the time of operation is of advantage. An accurate microscopic diagnosis will give the operator courage to do a radical operation if necessary or will give him confidence in the less radical procedure. However, it has not been our practice routinely to do a resection of the jaw in dealing with the ameloblastoma. The more accessible lesions are first removed locally. The patient is

warned that repeated postoperative examinations will be necessary over a long period of time to discover possible extension of the tumor. In many instances this local excision is adequate and not deforming. In other instances (1) where a large portion of the mandible is involved, (2) where the tumor has definitely invaded beyond the confines of bone, or (3) where inaccessible areas of the mandible are involved so that local excision could not be adequate, resection of the mandible is the primary treatment of choice (Figs. 2 and 3). Sometimes the choice of whether to do a local excision or radical resection must be made only after surgical exposure of the area of involvement. Especially is this true where the surgical approach is from the outside through the neck. In almost every instance where the surgical approach is from the inside of the mouth, local removal is done.

Intraoral Approach.—Having made the decision to do a conservative local removal rather than a resection of the mandible, adequate exposure must be gained by freeing overlying soft tissues and attached mucoperiosteum over the complete extent of the tumor. Following this, overlying bone should be removed adequately, so as to give a complete exposure of the underlying tumor and to permit partial obliteration of the cavity which results from the removal of the tumor. This obliteration is accomplished by collapse of soft tissue into the cavity. It is preferable not to rupture the capsule of the tumor itself during the operation. In many instances of the less loculated type of tumor the growth may be removed completely and without spillage. Under these circumstances there is less possibility of continued growth of the tumor.

Many of the multilocular tumors have irregular outlines and crevices in the bone at which point the tumor is usually broken into during the process of removal. One should never start out to do a curettement removal of this type of growth. It is much better to attempt total enucleation where possible. In multilocular or more invasive tumors one can become involved in a piecemeal removal of the tumor, however, and a certain amount of curettement will be necessary. Under these circumstances it is obvious that small fragments of growth will be left behind and necessitate removal at a later date.

To prevent this difficulty, cauterization of the cavity has been recommended. This cauterization may be either chemical or thermal. In general, the criticism of chemical cauterizing agents is that one cannot accurately determine the depth of penetration of the chemical so that cauterization tends to be either too great or too little in extent. The extent to which penetration goes and destruction is brought about with the thermal agent is better regulated than with the chemical. If the ameloblastoma has not been removed completely, it has been our practice to use the electrosurgical unit or Bovie knife with the coagulating current and to go over the exposed bone surface. Heavier cauterization is done in areas of greater contamination with tumor tissue. The disadvantage of this procedure is that a certain amount of bone will be devitalized and sequestration will occur. In the meantime, the presence of the devitalized bone will delay healing. However, if the tumor and its immediate bony confines have been completely removed or destroyed, cure is almost sure to result.

Extraoral Approach.—The external approach for both resection of the mandible and for local excision of the growth is the same. This incision should parallel the natural fold in the skin of the neck. It should be made underneath the mandible and should be adequate in extent to give complete exposure. The course of the lowest branch of the facial nerve in the neck should be remembered. This nerve can be preserved if the approach is carefully planned. The incision should be marked on the skin with the head in a normal position and should be 2 cm. below the angle of the mandible. A flap containing skin, subcutaneous tissue, and platysma is then elevated. This includes the lower pole of the parotid gland and soft tissues anterior to it. Unless the lower pole of the parotid is elevated with this tissue the facial nerve must inevitably be severed. The entire ramus as fell as the angle and most of the body may be thus exposed. Having exposed the area of the tumor, removal may be done in one of three ways. The procedure of local removal by shelling out the tumor and applying the cauterizing agent may be followed in certain cases. In other instances it is possible to do a partial block resection of the mandible; the Albee saw is used to remove the tumor and a containing block of bone, but a bridge of bone is left to maintain the continuity of the mandible. This is rarely feasible. The third method is to resect the mandible (Figs. 2 and 3).

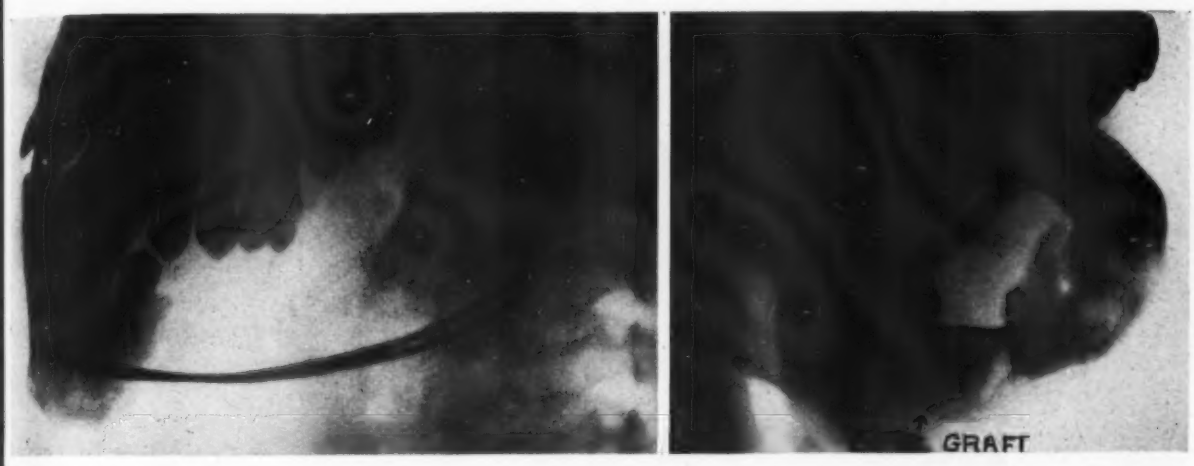
TABLE III. VARIOUS APPLIANCES USED TO CONTROL FRAGMENTS OF MANDIBLE AFTER RESECTION

I
Appliances which utilize teeth (intraoral): posterior fragment contains teeth
*a. Interdental wires
*b. Arch bars
*c. Swaged metal splint (with or without jackscrew)
d. Splint on teeth with dependent part separating bone
II
Appliances which utilize bone and teeth (intraoral and extraoral): short edentulous posterior fragment
a. Wire teeth in occlusion and use modeling compound in molar area to keep posterior fragment down
*b. Arch bar or splint with posterior fork to keep posterior fragment down
*c. Arch bar or splint on mandible with posterior portion accurately fitted to junction of body and anterior border of ramus (Herpin splint with or without jackscrew)
*d. As in *c* except arch bar or splint applied to maxilla with interdental wiring
e. Arch bar or splint with posterior part attached:
1. Directly to bone
2. To bone screw
f. Wire passed through drill hole near angle of mandible and attached posteriorly to head cap and wire rest of jaw in occlusion
III
Appliances which utilize bone (intraoral and extraoral): Number of teeth and size of posterior fragment not necessarily a consideration
†a. Bone screws (and bar) inserted with each fragment (Stader, Berry, Griffin, etc.)
1. Intraoral
2. Extraoral (preferred)
b. Kirschner wires (modified) passed through fragments
c. Bar passed through drill holes in end of each fragment
*d. Open bite splint attached with circumferential wires

*Also controls fragments adequately for insertion of bone graft.

†Not ideal for bone graft because of local osteomyelitis around bone screws and subsequent loss of stability of splint.

The site of resection of the mandible has usually been determined beforehand by studying the roentgenogram. If possible some portion of periosteum is left intact to bridge the gap. All bleeding is controlled. Where possible, mucosa is sutured so as to screen off the mouth from the neck portion of the wound, thus giving less contamination. The neck wound is closed accurately with a drain inserted.



A. *B.*

Fig. 7.—*A*, Lateral roentgenogram of mandible after subperiosteal resection showing one method by which position of mandibular fragments can be maintained prior to insertion of bone graft. A stainless steel bar was inserted through drill holes in the bone. It has now been present for over eight months, the wound has healed and bony union has occurred. In another instance a stainless steel bar was extruded after four years without suppuration. *B*, Lateral roentgenogram of the mandible after resection, stabilization of mandibular fragments, and insertion of bone graft. The opposite side of the jaw has been wired in occlusion and an attached splint is holding the posterior fragment in position.

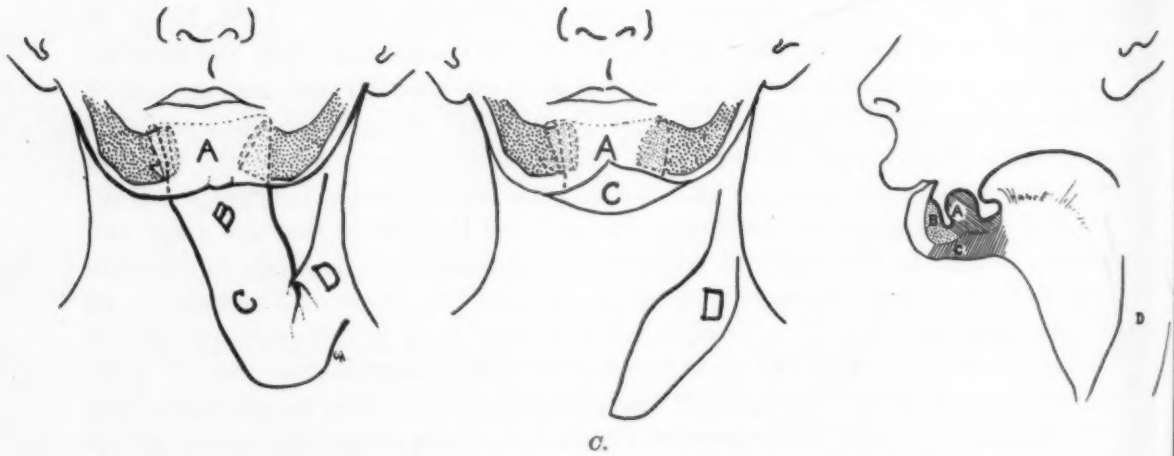
Maintenance of Jaw Fragments.—Wherever a resection is done some provision must be made to maintain the remaining fragments of the mandible in as near proper position as possible prior to bone graft repair (Table III). If there has been disarticulation in removal of the ramus, then only the anterior fragment must be considered, and in this case the simplest and best method of maintaining position is to wire the remaining teeth in occlusion until complete soft tissue healing has taken place. This same procedure may be the simplest also where there are teeth on both the anterior and posterior fragments. However, if the resection has been near the angle of the jaw so that the angle and ramus remain as the posterior segment, some means must be taken to maintain not only the space between fragments but also the posterior fragment in its proper position (Fig. 7). Various methods have been used to control the posterior fragment (Table III). It is essential that this space retention be maintained until there is complete soft tissue healing. Otherwise, as soft tissue healing occurs there will be progressive dislocation of the fragments by the pull of the new forming scar tissue and the various muscles. This dislocation will interfere with later repair by bone graft. This condition is especially common where mucosa has been removed and a granulating wound has been left inside the mouth to heal by secondary intention.

Reconstruction of the Defect.—Many patients have apparently been content to continue without having the defect in the mandible repaired. In some instances it has been possible for them to wear dentures. Unquestionably, the patient is more comfortable and his situation is more normal if a repair of the resected area of the mandible can be obtained with bony union. A number of methods may be employed but in general a free bone graft is the most desirable procedure.

In order to carry out the procedure of a free bone graft satisfactorily, certain things are necessary. The fragments of the mandible which remain must be free enough so that they can be not only placed but also maintained in their



A. *B.*



C.

Fig. 8.—*A*, Preoperative (taken on Nov. 2, 1944), and, *B*, postoperative (taken on Feb. 21, 1945) photographs of patient who had lost the anterior portion of his mandible from bicuspid to bicuspid area. There had also been loss of soft tissue. The scar tissue binding the anterior mandibular fragments was released. A neck chest flap was inserted to give added soft tissue. This same flap was used to line the buccal sulcus and cover lower border of chin. A bone graft has been inserted subsequently. *C*, Diagrammatic representation of source and use of flap tissue in *B*.

proper position during the time that the bone graft is healing in place (Fig. 7, *B*). It is extremely important that the covering soft tissue be adequate in quality and amount to give good covering and nourishment to a bone graft. If the overlying tissue has been damaged to the point that it is scarred and tight, the graft is likely to be extruded. If it is necessary to dissect dense scar tissue from between the ends of the bone before they can be restored to their proper position, then this must be done prior to the insertion of the bone graft to prevent mouth contamination. In certain instances it may even be necessary to apply additional soft tissue by means of flaps (Fig. 8) to replace scar or to supplement inadequate covering material which is present. This is done prior to the insertion of the bone graft. This is well illustrated in one patient (Fig. 8) who had lost two bone grafts because they were placed in dense scar tissue.



A. *B.* *C.*

Fig. 9.—*A*, Preoperative (taken on Nov. 2, 1942) and, *B* and *C*, postoperative (taken on Dec. 20, 1943) photographs of a patient who had lost the entire left side of the mandible. The bony and cartilaginous portions of a rib were utilized. The bony portion was attached to the remaining mandible and the cartilaginous portion was inserted into the temporomandibular fossa. Note the increased fullness of the left side of the face in *B*. *C*, The dotted line indicates the position of the graft, the stippled area represents the bony portion of the graft while the clear area represents the cartilaginous portion of the graft.

The bone graft may be either massive, using the half or full thickness of the rib or a section of the ileum, or it may be thin, as in the case of the osteoperiosteal graft taken from the tibia. This type of graft may be applied in several thicknesses. Regardless of the type of graft used, it is desirable to overlay the ends of the mandible with an inch or so of the bone graft where possible, making the bone graft 2 inches longer than the defect. In some instances the portion of the graft which overlaps the ends of the mandible may be thin while the portion between the ends is thicker. Sometimes a T-shaped graft may be employed, the thick arm of the T projecting in between the ends of the mandible. In all instances, the bone grafts should be in as close contact with the mandible as possible and anchored to soft tissues or even to the bone itself with silk or fine tantalum wire sutures.

Where there has been a disarticulation of one side of the mandible, a rib graft containing cartilage and bone can be utilized. The cartilage end can be fitted into the temporomandibular fossa and the bony end attached to the remaining mandible (Fig. 9).

SUMMARY AND CONCLUSIONS

The ameloblastoma arises from the enamel organ during a particular phase of tooth development, namely, morphodifferentiation. Because the cells from which this tumor arises are not as yet differentiated into ameloblasts, the term preameloblastoma is more accurate.

This tumor is found most frequently near the angle of the mandible in young adults. Although the clinical history and roentgenographic findings are an aid, the final diagnosis depends upon microscopic examination.

It is slow growing and unless completely removed will continue to grow (rather than recur). The ameloblastoma seldom becomes truly malignant.

Curettement, cauterization with drugs, and radiation are inadequate therapeutic measures. The lesion should be (1) completely enucleated if unilocular, (2) cauterized by heat if not too large and multilocular, or (3) if extensive, resected, including a small amount of normal bone. These are the best methods of treatment because they give the greatest assurance of no tumor being left.

The surgical approach and procedure for various ameloblastomas and the necessary subsequent reconstruction surgery have been considered.

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A TREATISE ON HARELIP AND CLEFT PALATE

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CLEFTS are invariably congenital, but may be also acquired through injury or disease.

ETIOLOGY

Congenital clefts and fissures are the result of arrested development. Fusion of the globular and maxillary, and the palatal processes in the embryo is completed about the end of the third month of fetal life, so that interference with development takes place prior to that time and continues. Many causes have been given for arrested development, but none have been found to sufficiently explain its occurrence in all cases.

The most important causes are faulty nutrition, heredity, and maternal impression. The influence of faulty nutrition has not been clearly proved in the human race, as women who received the most liberal and nourishing diet during the entire period of gestation have frequently given birth to children with clefts. On the contrary, women who existed on the most meager diet and on one or two varieties of food during the entire period of gestation have given birth to perfectly developed children. In animals, especially the lion, the influence of faulty nutrition has been repeatedly proved in the zoological gardens of London and Dublin. The process of growth and development depends upon the formation of loops of blood vessels in the embryonic tissue, which become organized tissue. In defective vascular supply may be found a probable cause of arrested development and this would explain its occurrence in early fetal life and at the terminal distribution of the vascular supply. The vascular supply to the right half of the head is greater than to the left. This probably accounts for the most frequent occurrence of the clefts of the lips and alveolar process on the left side.

The frequent occurrence of clefts in the descendants of those who had clefts and in members of the same family points strongly to heredity. In many cases the occurrence of the affliction does not take place until the second, third, or fourth generation.

At present the influence of maternal impressions cannot be absolutely denied, but the mother will state positively, that she had not the maternal impressions during gestation, and in many cases where strong maternal impressions existed, the offspring was perfectly developed. Many eminent obstetricians have admitted the possibility of maternal impression being the cause.

Clefts may be the result of syphilis. Such clefts should rarely, if ever, be operated upon, as union seldom takes place and the operation frequently results in increased destruction of tissue.

Based on lectures on Oral Surgery by the late William J. Roe, M.D., D.D.S., Pennsylvania College of Dental Surgery, and Jefferson Medical College, and the Oral Surgery Clinics at The Philadelphia (Blockley's) Hospital.

CLEFTS OF THE LIPS

Clefts of the lower lip are very rare and involve the median line and may extend through the chin and mandible. Clefts of the upper lip are quite common and may be of any extent, from a simple notch in the lower border to complete fissure extending into the floor of the nose. Here clefts are rarely situated in the median line, but a little to either side, most frequently to the left. Unilateral cleft occurring on one side, and bilateral clefts occurring on both sides may be found. Complete clefts extend through the lip into the nose. Incomplete clefts extend only into the base of the lip.

Prognosis.—An infant with complete cleft of the lip is seldom able to suckle; therefore, its nourishment is greatly interfered with as it is often difficult or even impossible to nourish it properly by artificial feeding, and many children die from this cause.

Treatment.—Operative treatment is here clearly indicated, as little if any reparative process takes place during growth. The time at which the operation should be performed is of the greatest importance. The consensus of opinion is to perform the operation as early as it can be done with safety. In strong vigorous infants, the operation can be performed in from three to six weeks after birth, but it is better to defer it until the period between the third month and the beginning of dentition. In delicate infants or those suffering from pulmonary or gastrointestinal disorders, the operation should be postponed until improvement has taken place.

Operation.—(Cheiloplasty, chielorrhaphy.) A general anesthetic of choice is administered. The attachment of the lip to the alveolar process is freely separated to allow easy approximation and to prevent traction. The deputation of the margins of the cleft is freely made with a bistoury or scissors, being careful to obtain straight cutaneous margins and broad approximal surfaces. The assistant controls the hemorrhage by applying pressure upon the vessels in the lips, holding it between his thumb and fingers. Two or three sutures of silkworm-gut or silver wire are carried through the skin and lip down to the mucous membrane. The first one should be introduced at the vermilion border, taking care to make accurate approximation. The margins of the mucous membrane and skin can be further approximated, if necessary, with fine silk sutures. Harelip pins should seldom be applied as a much larger stitch wound follows their use than with sutures. The sutures can be removed in from five to eight days. Bilateral clefts can be closed in the same manner and at the same time, but one side may be left for a subsequent operation. If the intervening portion, which, when present, is connected with the nasal septum, is absent or too small to be utilized, lateral horizontal incisions extending from the ala of the nose outward will allow the lips to be brought together.

CLEFTS OF THE ALVEOLAR PROCESS

Cleft of the alveolar process is practically never present, except when associated with cleft of the lip and cleft of the hard and soft palate it involves the suture upon one or both sides of the premaxillary bones, most frequently

upon the left side. In unilateral cleft the premaxillary bone is projecting forward, and to the sides opposite the cleft. In bilateral cleft it projects far forward, and is attached to the nasal septum.

Treatment.—When associated with cleft of the lip, it should be operated on previous to or during the operation for cleft of the lip.

Operation.—The margins of the cleft are denuded, and the bone is restored to position by fracturing or chiseling its attachment to the maxillary bone or nasal septum, and secured in position by wire sutures carried through the alveolar process. In some cases the pressure of the lip will suffice to hold the bone in position. In cases of bilateral cleft the bone is sometimes so rudimentary that it is better excised. When associated with complete cleft of the lip, hard and soft palate, it is usually left to be repaired with the hard and soft palate, except where it projects and interferes with the closure of the lip.

CLEFT OF THE SOFT PALATE

Cleft of the soft palate is always situated in the median line and may vary in extent from a simple bifid uvula to a complete cleft extending to the hard palate.

Treatment.—If the cleft is extensive enough to interfere with the function of the palate, it should be operated upon. Incomplete clefts will lessen considerably in extent during growth, but seldom sufficiently to restore the function of the palate.

Operation.—(Staphylorrhaphy.) If the operation is performed and the palate restored before the child learns to talk with the cleft, the voice is seldom interfered with. If the operation is delayed, it may require months, or years, of careful training to correct the acquired nasal voice, and frequently it continues throughout life. The objection to early operation is the comparatively high mortality. The operation for the repair of a palatal or labial cleft is seldom, if ever, justifiable, if the mortality rate is above that which belongs to the calamities of surgery. The operation can be performed after the sixth month, but is more easily and safely accomplished between the second and fourth year. A general anesthetic is given young children; older children and adults can frequently have it done under local anesthesia. The margins of the cleft are denuded, sutures preferably of silver wire are introduced from the posterior surface and placed from ¼ to ⅓ inch apart. When the palate is brought together, the sutures are twisted or tied upon the palatine surface. Silk or catgut sutures are frequently used to unite the uvula. If the palate is very tense and cannot be approximated, the tendons of the tensor palati muscles should be cut where they pass around the hamular process.

CLEFT OF THE HARD PALATE

Clefts of the hard palate vary in extent from a mere notch in the posterior border of the horizontal plates of the palatine bones to complete cleft, involving the palatine process of the posterior maxilla and the maxillopremaxillary sutures. The fissure is always situated in the median line of the hard palate, but extends

more often to the left than the right side. Therefore, in these cases the nasal septum remains attached to the right side. Where the fissure is very wide, the nasal septum is free in its entire extent. These cases are spoken of as double clefts. Partial clefts are most frequently associated with clefts of the soft palate. Complete clefts are associated with clefts of the soft palate and lips.

Treatment.—The mechanical and surgical arts vie with each other in the treatment of this condition. Many leading authorities recommend wearing an artificial obturator or vellum. Many others recommend operation in all cases. Most gratifying results are often obtained from wearing the obturator and vellum, and the mortality is nil. Against this, however, one must consider the moral effects of the consciousness of an existing deformity, the frequent difficulty and annoyance of wearing an artificial appliance, and the irritation which it produces. The fact that it cannot be worn in early childhood to prevent the child from acquiring the nasal voice is a great handicap.

Operation (Uranoplasty).—The advantages of surgical treatment are many. The operation can be done in early childhood before the child has learned to talk, or at the period at which the voice can be properly cultivated. The moral perversion which is so frequent in these cases is usually prevented, for the children grow up without realizing that they have any defect, and the life-long annoyance and irritation of wearing an artificial appliance is avoided. Against these advantages, however, is to be considered the mortality attending surgical treatment, and the too frequent failure to obtain union. The mortality of various operators ranges from 1 to 15 per cent. It lessens with the increased age of the child. In strong healthy children the operation can be performed safely in the period from six months to two years. The best and safest period in the majority of cases, however, is between the second and fourth year. In delicate children the operation may have to be deferred until after puberty. It is now quite possible to close any cleft by one, or a combination, of the three following operations.

Fergusson's Operation for the Cleft of the Hard Palate.—The margins are first denuded, the holes are drilled through the palatine process from ¼ to ⅓ inch apart, and about ¼ inch from the margin of the cleft. Through these the sutures are introduced from the nasal to the palatine surface, but not tied. Lateral incisions are now made through the mucous membrane and periosteum, a sufficient distance external to the rows of the suture so as not to disturb them. The bone is divided by means of a chisel, beginning with the horizontal plate of the palate and ending opposite to, or a little in front of, the apex of the cleft. The flaps can be approximated and any undue tension overcome by using the chisel as a lever, after which the sutures are tied and the lateral wounds packed with iodoform gauze. It is best to repair the soft palate at the same time, but if the patient is doing badly it can be deferred to a subsequent time.

Lagenbeck's Operation for the Cleft in the Hard Palate.—The margins of the cleft are denuded; two lateral incisions parallel to the cleft are made through the mucous membrane and periosteum overlying the alveolar process beginning in the soft palate and extending forward to the incisor region. By means of the periosteal elevator, the mucous membrane and periosteum are detached from the bone and the soft palate is detached from the posterior border of the hori-

zontal plate of the palatine bone. The inner margin of these flaps, together with soft palate, are brought together and sutured, thus closing the cleft. Lagenbeck rarely sutured the soft palate at this operation, but it is usually best to close the cleft in the soft palate at the same time. Bone frequently forms from the periosteum and strengthens the flap.

Brophy's Operation for the Cleft in the Hard Palate.—The Brophy operation for the cleft is indicated in complete cleft which is too wide to close by either of the preceding operations. In some instances the maxillary bones are abnormally separated, and in such cases Brophy's method is admirably adapted to repair the cleft and restore the normal contour. Silver wires are passed horizontally through the maxillary bones above the hard palate. The anterior one in front of the malar process and the other joint posterior to it. The ends of the sutures are passed through two disks of lead placed against the malar process over which the wires are twisted. The margins of the cleft are denuded and the maxillary bones pressed together and the wires tightened. This operation should be performed in the early weeks or months of infancy at which time the bones will be readily pressed together. If resistance is experienced, the malar process on either side is detached by the scalpel or chisel. Additional sutures in the margins of the cleft can be introduced if required for better approximation, or a subsequent operation can be done at a later period to repair the soft palate or any part of the cleft remaining in the hard palate. Aluminum disks may be used instead of the lead disks to avoid the possibility of lead poisoning. Brophy claimed that he has not had any evidence of lead poisoning from the use of lead disks.

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OXYGEN-WANT IN GENERAL ANESTHESIA

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WHEN the body tissues fail to receive an adequate supply of oxygen during the process of the interchange of gases we call "respiration," a condition of anoxia ensues. This state may be induced by various conditions, chief among which are: low oxygen tension in the inspired atmosphere, a poisoning of the inspired air by some inert gas, by high altitudes, and by various physiologic disturbances arising from asthmatic or pneumococcic infections.

For the dental anesthetist, the problem of anoxia resolves itself into a desire to be on the alert to prevent displacement of the essential oxygen content of the blood and body tissues by other gases which may hinder the normal functioning of heart, brain, and kidneys. This point is important to bear in mind when discussing a gas such as nitrous oxide, because those who defend it speak of it most tenderly as a harmless agent which undergoes no change during inspiration and expiration. It is my aim to focus the attention of the dental profession upon the potentialities of danger which lurk in the inhalation of nitrous oxide, not per se, but because, in its inhalation *to induce anesthesia,* one is compelled to reduce the oxygen quotient of the inhalant gas volume below the 20 per cent oxygen level that our bodies require, *asleep or awake.* I would have no fault to find with nitrous oxide as an anesthetic agent if it were possible to induce narcosis without oxygen deprivation. *It is because anoxia is always present when nitrous oxide is inhaled for complete anesthesia* that I feel the need very urgent for the profession to make an honest and courageous reappraisal of its attitude toward this agent. Certain changes in procedure must be considered. They are changes that are simple, and are called for in the interest of the safety of the patient who entrusts his life to us. Voices are being raised in various quarters, feeble as yet, but growing in volume daily. They must be heeded. The challenge of safety in general anesthesia must be met squarely by the profession. The time is past when asphyxia may be used and advocated as a legitimate concomitant to the induction of anesthesia with nitrous oxide.

Dr. Cyril Courville, of Los Angeles, California, has published a text called *The Untoward Effects of Nitrous Oxide*† which I consider an epoch-making contribution to the science of anesthesiology. In the foreword of this volume, the late Dr. Yandell Henderson, Professor of Applied Physiology at Yale University, wrote:

"Dr. Courville has rendered a service of high value by demonstrating that nitrous oxide, *even for short anesthesias,* is far from deserving its reputation for safety; and that when employed for prolonged anesthesia it often involves degrees of anoxia sufficient to injure the brain, and even to result in delayed asphyxial death."

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†Pacific Publishing Co., Mountain View, Calif.

"Asphyxia," says Dr. Henderson further on in the same text, "is one of the hazards inherent in all forms of anesthesia. But with no other anesthetic is this hazard so notable as with nitrous oxide. It is even believed that full anesthesia cannot be induced with nitrous oxide without an accessory partial asphyxia. Nevertheless, for many years nitrous oxide was considered to be the safest of all anesthetics."

Dr. Alvin Barach, Professor of Medicine at Columbia University, has written me a personal communication in which he says: "You are quite right that nitrous oxide anesthesia should never be given with a concentration of less than twenty per cent oxygen, since we know how sensitive the brain is to anoxia." Dr. Barach says, in his excellent text, *Inhalation Therapy*,* regarding the need for rich oxygen during childbirth anesthesia:

"Fetal anoxia in the period before delivery is the cause of many still-births, and also of death in infants who die shortly after delivery, due to cerebral pathology inflicted by severe anoxia during delivery. Fetal anoxia may be caused by interference with the maternal supply of oxygen and also by factors which prevent the fetus from receiving oxygen. *In the first category is included the low oxygen concentration present in certain anesthetic mixtures of nitrous oxide.*† Although nitrous oxide is of inestimable value in preventing labor pain, it may be administered with a low concentration of oxygen so continuously that the mother experiences anoxia for a prolonged period."

Thus, Dr. Frederic Schreiber, of Detroit, Michigan, Professor of Neurological Surgery at Wayne College of Medicine, has published a report covering an investigation of cerebral anoxia at birth as cause of mental deficiency: "Alterations in the tissue architecture of the brain as a result of diminished oxygen supply or insufficient oxygen consumption may cause permanent retrograde changes in the intellectual development of the individual subjected to these destructive effects. Neural cells cannot tolerate severe anoxia for more than a few minutes without permanent alterations in the tissue structure. Such an anoxic insult may be survived by the individual since the cortical cells are more quickly damaged by oxygen-lack than are the cell groups housing the vital centers. In that case it appears reasonable that 'devastation areas' which may occur mainly in the frontal lobes secondary to anoxia will find clinical expression in deterioration of intelligence." Dr. Schreiber examined the birth records of several hundred mentally deficient children under 10 years of age, and found an amazingly impressive number to have had a history of apnea at birth.

Reports from various sources point to a definite marked increase in morbidity and mortality from nitrous oxide administered without atmospheric oxygen sufficient for the body needs. The fear of any untoward result from an asphyxial episode with nitrous oxide continues to be minimized by the devout adherents of this agent.

Drs. Rovenstine and Barach, in their contribution to the *Journal of Anesthesiology* for September, 1945, entitled, "The Hazard of Anoxia During Nitrous Oxide Anesthesia," say:

*J. B. Lippincott Co., Philadelphia, Pa.
†Italics mine.

"The mortality and morbidity following nitrous oxide anesthesia in hospital practice is apparently not high when determined from reported statistics. This may be true but there is much misleading information accumulated, since it is frequently the practice to consider as anesthetic deaths only those that occur in the operating room or soon thereafter. Moreover, in hospital practice, if there is evidence of asphyxia during nitrous oxide anesthesia, the usual practice is to add another agent which may then be held responsible for any untoward results. The mortality and morbidity statistics for nitrous oxide-oxygen anesthesia in office and clinic practice are difficult to obtain, since reports from these sources are infrequent and inadequate. If statistics are collected from the files of daily newspapers, the representatives of insurance companies, and the personal reports from confreres, anesthetists must admit it is not negligible. Newspapers usually publish reports when the subject has sufficient prominence to merit a 'story.' Since deaths do occur not infrequently during and as a result of nitrous oxide anesthesia, one must assume that technical errors are often committed, or the present approved principles for administering nitrous oxide are themselves at fault."

As an example of an untoward result from nitrous oxide anesthesia which never gets newspaper publicity, or is recorded in any record of statistics dealing with safety in general anesthesia, I have in mind a case of a hospital death brought to my attention, which very pertinently illustrates the above thought. A young man of 30 years had had a number of teeth removed under nitrous oxide in a dental office. The operation had lasted fifteen minutes. The patient was removed to the hospital in a delirious state of mind, never became rational, and died ten days later. Autopsy showed degenerative areas in the brain definitely ascribed to lack of oxygen during the administration of the nitrous oxide. The changes in the brain tissue were those typical of asphyxia. Unfortunately, not all such victims of asphyxial episodes terminate their earthly existence in this way. They are those with definite personality changes, who, while rational, go through life permanently crippled mentally, as for example, the case cited by Dr. Roy McClure of the University of Michigan:

"A student of the University of Michigan was leading her class in the particular group. She came to Detroit and had an impacted molar extracted. She went alone to the dentist. When she reached home that afternoon, her vision was blurred. She was taken to an ophthalmologist, and glasses were prescribed. In school her companions noticed no change in her, but the teachers did. Her work began to fall off, and before many months had passed, this girl failed completely in her work. She returned home and visited the same dentist to have an impacted molar on the other side removed. He said, 'Don't let anybody give you gas again, for you can't take it.' She had become blue; artificial respiration had been given."

While there seems to be a varying tolerance among individuals for low oxygen concentrations, the evidence seems to point to the extreme hazard of permitting, for the great majority of patients, an asphyxial anesthesia even for extremely short periods. A typical illustration of the effects of anoxia of short duration is seen in the routine training given to our flying men. In a communication from the commanding officer of one of our large training camps in this country there came the following message:

"As regards anesthesia the writer is presumably concerned with acute effects of anoxia. Every air crew trainee in the A.A.F. is exposed at least once to the acute effects of anoxia during his indoctrination procedure. It is fre-

quently carried to a state of unconsciousness. There is no evidence of any ill effects from this procedure on these trainees. Considerable numbers of fliers lose consciousness from the lack of oxygen during flights in high altitudes from many causes. There is no evidence of damage to the tissues of the heart, brain, or lungs, or any body function to those who recover consciousness. *On the other hand, deaths resulted without regaining consciousness.** Whether or not an individual will regain consciousness after anoxia will depend on the severity as well as on other less well understood factors."

This is, in some ways, a remarkable statement, because of its serious implications. It is inconceivable to me how one can speak of failure to recover consciousness on one hand, while making the observation that no tissue changes have been noted in those who do recover consciousness. Surely there must be some cases *in between the two zones,* cases as recited above, where the young woman recovered from the dental anesthetic but had lost her personality.

Wherever studies of brain tissue have been made in human beings, there has been definite corroboration of the findings of scientific investigators working with animals. Necrosis of the cortex has been shown in work with cats, pigs, and monkeys, by the deliberate production of anoxia. The work of Gildea and Cobb, and of Thorner and Lewy, for example, leaves no doubt of this fact. With human beings, the work of Courville, Schreiber, McClure, Hartman, Schendorf, and Schelling, and many others in the literature, is outstandingly convincing.

Thus far, this treatise has been confined practically to a consideration of the effects of anoxia on brain tissue. Let us consider briefly the cardiac manifestations associated with asphyxial concentrations of nitrous oxide and oxygen, or rather the *lack* of oxygen.

I have a communication from a prominent cardiologist as follows:

"Nitrous oxide is not a desirable anesthetic from the point of view of the heart, and I believe that it is apt to throw an undesirable burden even upon a healthy heart. The reason for this is the fact that the oxygen content of the mixture which the patient breathes is less than the normal amount in the atmosphere. This means a strain for any heart equivalent to a high altitude. In fact we use an anoxemia test (ten per cent oxygen and ninety per cent nitrogen) to bring out anginal pain and changes in the electrocardiogram in latent cases of coronary disease. Therefore, reducing the oxygen intake is a strain for the normal heart when you add the additional strains of anesthesia and surgical intervention. For a very brief anesthesia there is sufficient reserve in the average heart to take up this extra strain with no symptoms, but the subnormal heart might develop symptoms, and certainly should not be subjected to this strain. We do not know when we are dealing with a subnormal heart in many cases, as we use an anoxemia test precisely to discover this, and nitrous oxide anesthesia could in a way be considered roughly the equivalent of such an anoxemia test. However, if the nitrous oxide is handled expertly, such strain is kept down to a minimum, and the real danger is the fact that it is not always handled expertly. Therefore, cyanosis is common when it is used, and that certainly is evidence of undesirable strain upon the heart.

"Any anesthetic which can be used with extra amounts of oxygen, that is, a percentage of oxygen in excess of the average amount in the atmosphere is definitely desirable for all hearts, both normal and subnormal in capacity. For this reason, in certain surgical work in coronary disease we use a gas which permits 60 and 70 per cent or more of oxygen to be given with it. If you can give an oxygen-rich mixture of ethylene (amounting to 40 or 50 per cent oxygen)

*Italics mine.

you are certainly doing much to help any heart and materially reduce the strain of anesthesia.

"The routine use of nitrous oxide with the lesser percentage of oxygen is certainly undesirable as compared to another gas such as Ethylene with a higher percentage of oxygen."

Van Liere, in his work on *Anoxia* shows conclusively that cardiac output is increased when oxygen below atmospheric needs is reduced considerably. True, the very healthy heart will withstand this burden, but already handicapped ones will not. The load thus put on a poor heart may cause it to go into failure. Anesthetists may console themselves with the thought that the patient had already had a weakened heart prior to the anesthetic episode, but this is beside the point. My experience has shown me that, given a rich oxygen mixture with the anesthetic agent, and given a patient who is thoroughly cooperative, the weakened heart is subjected to no greater jeopardy than when local anesthesia is employed. I do not consider a heart disability contraindicative to the availability of general anesthesia unless there are gross symptoms of decompensation present. I have been successfully administering a rich oxygen general anesthesia, yes, an oxygen quotient far above that in the atmosphere, to individuals with a history of coronary occlusion, or marked hypertension. I do this as daily routine, with the utmost satisfaction to all concerned. The important thing to remember, and I repeat, is that such a patient be completely cooperative. We must avoid the sudden outpouring of adrenalin into the blood stream. Anesthesia for such handicapped individuals must never be forced; that is, pushed against the patient's desires. The moment such a patient evidences a severe distaste for the ordeal, it is my procedure to remove the inhaler at once. The anesthetist must be alert to distinguish between possible excitement symptoms and a desire by the patient to have the anesthetic discontinued. By so doing, an anesthetist may avoid much embarrassment.

There are many dental anesthetists who refuse a general anesthetic to known cardiacs. Dental schools teach likewise. This is unwarranted by clinical experience. The late Dr. James T. Gwathmey has stated on innumerable occasions in my presence and to me personally, that, any agent, local or general, which can induce anesthesia, can induce death. In other words, local anesthesia is not necessarily a guarantee against accident. Both agents and procedures have their mortality records. Dr. Lundy, Director of Anesthesia at the Mayo Clinic, is representative of those in the field of anesthesia who advocate general anesthesia for the handicapped heart. In his book, *Clinical Anesthesia,** Dr. Lundy says:

"It has been my experience that angina pectoris is not a complication which contraindicates a general anesthetic especially if oxygen also is administered."

The important precaution, you may note, is the oxygen. And that is my theme. I feel that a high oxygen quotient is the central theme in what I call the symphony of respiration. The agents employed are only secondary. The oxygen is the essential element, and the operator's technique must adapt itself to the oxygen demands at all times. The anesthetic agent may be increased or reduced as conditions indicate, but one should not cut down on the oxygen supply.

*W. B. Saunders Co., Philadelphia, Pa., 1942.

Any alterations in the oxygen flow should be upward to higher levels, never downward to levels below 20 per cent atmospheric content.

In an address before the American Society for the Advancement of General Anesthesia in Dentistry, Dr. James T. Gwathmey, who introduced me years ago to ethylene and to vinethene, declared:

"I think if we continue to go on nitrous oxide alone, we will still hear of deaths in the dental chair every once in a while, because it doesn't make any difference how often you give nitrous oxide and oxygen, there will come a time when some patient—and he won't necessarily be a day laborer or an alcoholic, he may be a white collar clerk—for some reason will not go under and *then you will try to push the nitrous oxide and you may push him a little too far and you will get a fatality.**

"*Now your remedy for such cases is to combine vinethene or ethylene with nitrous oxide and oxygen.** You will avoid much trouble and have the pleasure of going through a year's work without seeing the captious display in some paper—and they always have it—'*Death in the Dental Chair.*' *We want to avoid that if we possibly can.*"

It is important to bear in mind constantly the fact that oxygen restriction may induce the subconscious mind of a rugged individual to put up a struggle under anesthesia—a struggle for survival—that may be sufficient to set off ventricular fibrillation.

It has been my procedure for some time, to administer as high as 50 per cent of oxygen with my anesthetic gases. This I do on the theory that if it will help the heart and circulatory system to supply an excess of oxygen reserve, I am in no way jeopardizing the patient for whom the ordeal of undergoing an anesthetic experience may be a strain. While the body does not actually use up an excess of oxygen delivered to it, there is scientific evidence tending to establish the fact that cardiocirculatory function is sustained. My own experience has definitely proved to me that the incidence of nausea is reduced as a result of the high oxygen level of my anesthetic gases. Inhalation of 100 per cent oxygen has been used to control nausea and vomiting of seasickness according to Barach. Some dental anesthetists scoff at the idea of very high oxygen concentrations in an anesthetic inhalant volume but they have no physiologic basis other than blind prejudice for their opinion. It is usually difficult to bring about changes in procedures which have a history as old as nitrous oxide practice. But the findings of the physiologists must prevail and we would do well to heed men like Alvin Barach, who says:

"Recent studies on the dosage of oxygen therapy have a direct bearing on current medical practice. In order to raise the concentration of oxygen in the arterial blood to a level near that of the normal, the percentage of oxygen generally required has been found to be 50 per cent. However, in certain patients who suffer from severe anoxia, such as shock, higher concentrations have a definite advantage. Although continuous inhalation of pure oxygen has been known to produce edema of the lungs of small animals exposed to it for periods of three to five days, it has become evident, largely as a result of the investigations of Evans and of Boothby, that the human being may tolerate for at least two days and probably for four days the inhalation of pure oxygen without harmful effects."

*Italics mine.

Studies in oxygen therapy have revealed that when atmospheres containing 40 to 60 per cent oxygen are inhaled, the oxygen saturation of arterial blood is raised to or near normal in patients suffering from pneumonia or cardiac insufficiency. This statement of fact is made in response to questions raised in some quarters lest the delivery of oxygen in high percentages be injurious to the body functions. There is sufficient clinical evidence that the body requirements are extracted from the rich oxygen mixture under conditions which materially support the cardiovascular system. Postoperative oxygen therapy is a universally accepted therapeutic procedure today. Inhalation of a rich oxygen quotient in an anesthetic mixture offers equally positive assurance of supportive therapy.

The problem, then, is a simple one, if the profession will heed the warnings that are coming from the investigating mind of the clinician. "A study of the experimental lesion following deprivation of the brain of its normal supply of oxygen for any length of time makes it quite clear that the cerebral lesion after nitrous oxide anesthesia is *an asphyxial and not a toxic one,*" says Courville, the neurologist. "I think that nitrous oxide and oxygen per se is one of the most dangerous anesthetics that we have," said James T. Gwathmey, anesthetist, shortly before he passed away last year. "Nitrous oxide and oxygen can be improved, and we shall improve it, but how can we do it? We must stop giving nitrous oxide and oxygen alone." "You must tell the profession," he wrote me in January of 1944, "the very great difference between an asphyxial anesthetic, such as nitrous oxide and oxygen alone, and one that takes advantage of all the good qualities of nitrous oxide and oxygen as an introductory and then supplementing with ethylene, thus securing a safe plentiful oxygen anesthesia for the anesthetist to work with as long as he wishes. You have an anesthetic that is so much safer than the usual nitrous oxide system followed by the dental profession that there is really no comparison."

We owe a duty to society to heed the message of caution. To fail to amend our anesthesia procedures in the interest of safety is unwarranted. We have a challenge that merits logical debate. It is going to be difficult to change the attitude of the profession toward an agent about which a century-old tradition has been built. But it must be done.

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APPLIANCES TO CORRECT RESECTED OR MISSING PORTIONS OF THE MANDIBLE

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THE prosthodontic phase of treatment of patients who have sustained the loss of portions of their mandible is dependent on the conditions and indications that prevail upon presentation. One of several appliances may be constructed, equal emphasis being placed on immediate requirements and on future treatment. The primary goal is to maintain or re-establish adequate functional occlusion and preserve or restore maximum possible esthetics.

Segments of the mandible may be lost as a result of one or more causes, which would include trauma, osteomyelitis, cancer, radionecrosis, and surgery. The extent of the loss will vary as will the location, and these factors in conjunction with masticatory muscle pull and scar formation will affect the design of the prosthesis to be constructed to restore some degree of function and facial contour in the rehabilitation procedure.

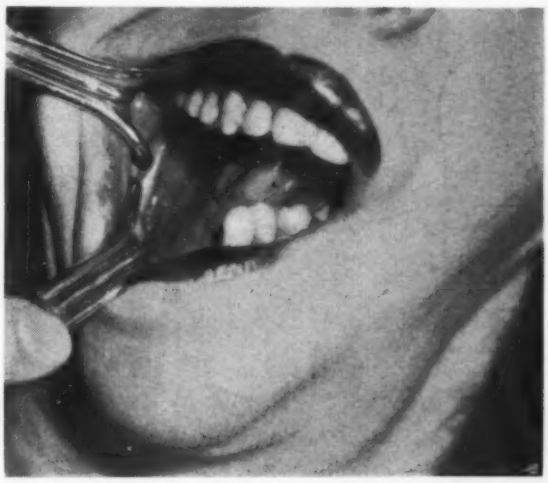


Fig. 1.—Patient with right ascending and horizontal rami surgically removed, showing remaining fragment drawn toward midline.

Loss of function is dependent on the amount of mandible lost and the former position of the lost fragment, not only because the number of teeth missing may offer a problem in replacement and stabilization, but also because unilateral cicatricial tissue traction on the defective side in certain larger resections or losses, plus muscle action on the normal side, will tend to draw the remaining horizontal fragment toward the midline (Fig. 1). Another effect that must be considered is the pulling of the remaining ascending ramus fragment out of position. Also, the loss of supporting bone may cause the collapse of associated soft tissue with resultant affected facial contour.

From the School of Dental and Oral Surgery, The Faculty of Medicine, Columbia University.

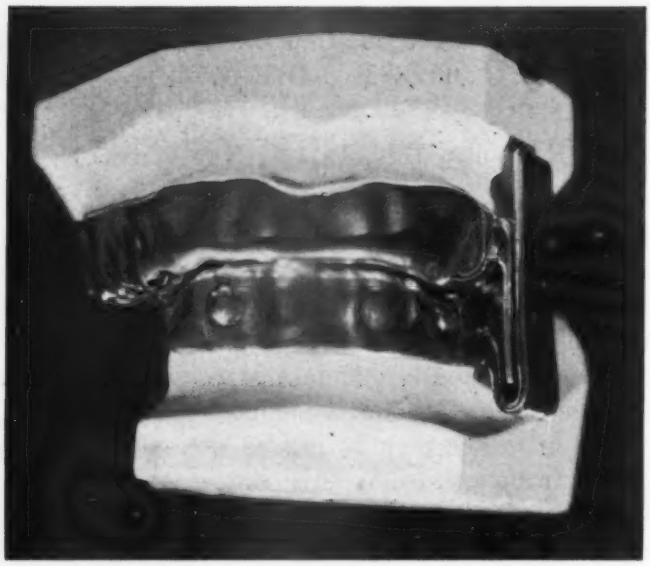


Fig. 2.—Cast sterling silver cap splints with buccal flanges.

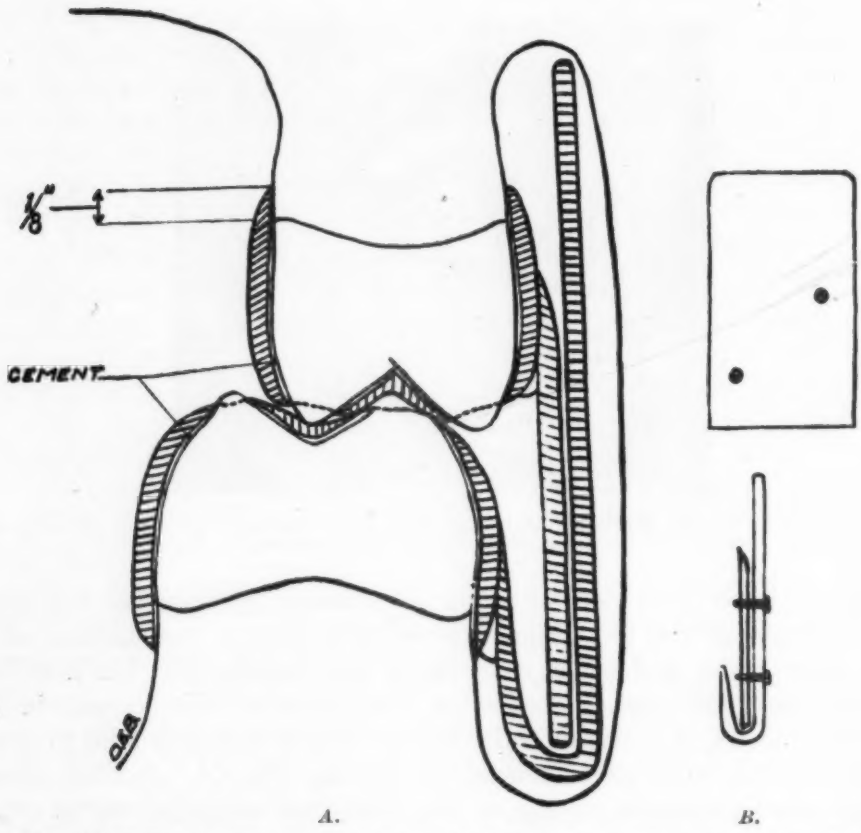


Fig. 3.—Diagram drawing (cross section) illustrating buccal flanges with locking device. *A*, Buccal flanges soldered to cast cap splint. *B*, Screws to lock splints through flanges.

CORRECTIVE APPLIANCES FOR UNILATERAL LOSS OF A JAW SEGMENT

Corrective appliances for unilateral losses may be in the nature of temporary or permanent prostheses. The former would include, for example, an upper and lower cast sterling silver cap splint with an adequate flange (whose upper and lower limits are the mucobuccal folds) soldered to each splint only on the uninjured side, to be used in cases where the ascending ramus is absent on the defective side (Fig. 2). Holes are cut through both flanges in the same horizontal plane, but each group being at a different point. These holes may be threaded so as to accommodate a screw, or may be left unthreaded to allow passage of a wire (Fig. 3). In either case, the screws or the wire will immobilize the upper and lower jaws when the two flanges are locked together. This would enable the surgeon to insert a bone or cartilage graft in suitable cases, allow it to unite and heal in its new site; and then, when the screws or wire are removed, it will make it possible for the patient to use the jaws in an adequate hinge movement without putting too great a stress on the new graft. Another form of prosthesis that can be used in certain types of these cases is the sectional splint[2] with an auxiliary flange on the side opposite the lost segment (Fig. 4).

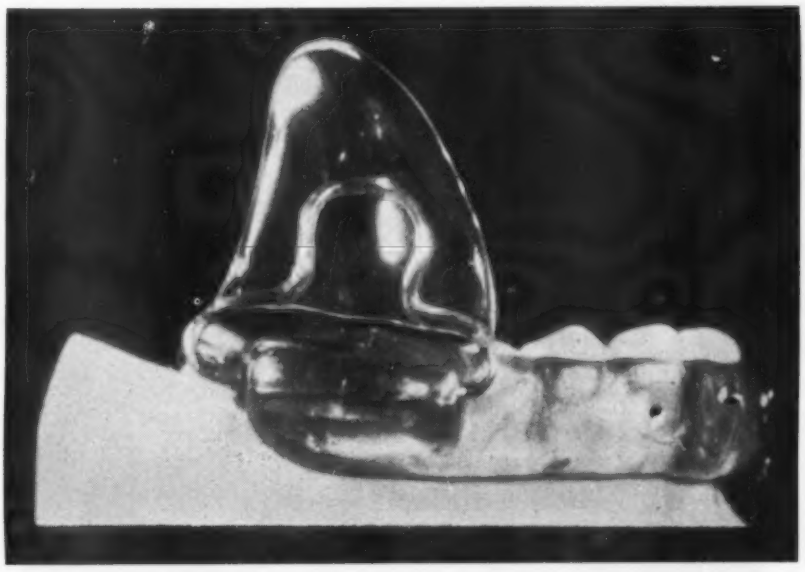


Fig. 4.—Buccal flange attached to sectional acrylic splint to maintain normal alignment of the mandible in loss of ramus cases.

It is used on the lower jaw, when the flange will contact a portion of a denture in the upper jaw, as direct contact with tissue would most likely injure the latter. Deviation of the remaining mandibular fragment is thus prevented. The flange may be locked to the denture, where necessary, by means of a pin or screw.

CORRECTIVE APPLIANCE FOR LATERAL SEGMENT OF HORIZONTAL RAMUS

Sometimes a portion of the horizontal ramus near or including the angle will be missing without loss of the ascending ramus. The latter will usually be drawn upward and inward. Consequently a prosthesis must be constructed

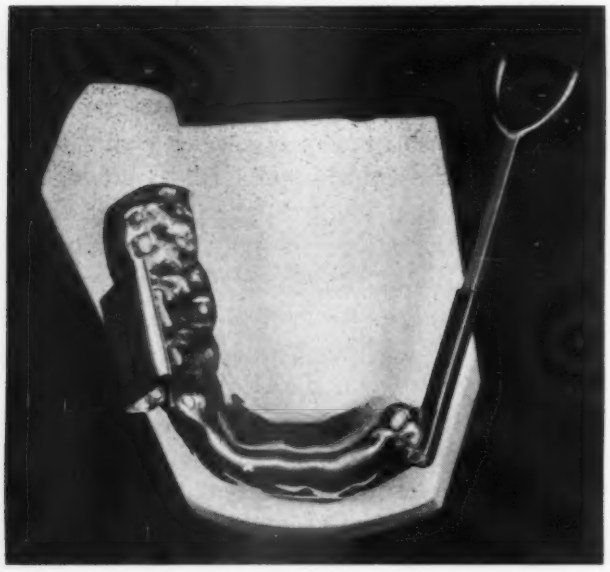


Fig. 5.—Auxiliary prong attachment on cast mandibular splint to immobilize ascending ramus in partial loss of horizontal ramus. Bar is locked by means of a pin through the end of the tube.

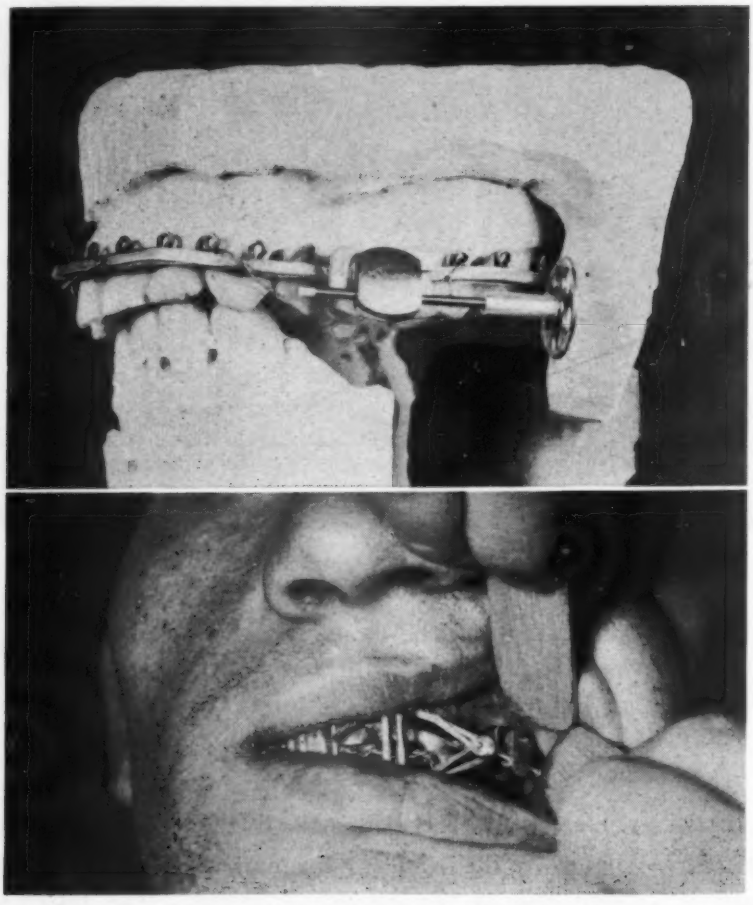


Fig. 6.—Adjustable bar maxillary splint designed by Dr. Roger Gerry for immobilizing ascending ramus. (Courtesy of Dr. Gerry.)

to reduce and immobilize it in its normal position. One type of applicable auxiliary appliance would be a 12 gauge square wire with a double-pronged round wire soldered to one end. The prongs are pushed directly into the correctly reduced ascending ramus. The square wire fits into a 12 gauge square tube that is soldered to a cast cap mandibular splint. The wire is locked in position by means of a pin through the tube. This appliance is used in conjunction with the double flanges on the normal side (Fig 5). Another satisfactory prosthesis in dentulous cases would be an adjustable bar attached to the maxillary teeth or ridge by means of a splint. The bar has a bulk of acrylic, vulcanite, or even compound attached to its free end, and which is contoured to fit against the anterior border of the ascending ramus. Thus, with the latter held in position and the jaws fixed by means of intermaxillary elastics or wiring, the surgeon has free access for the insertion of a bone graft (Fig. 6).

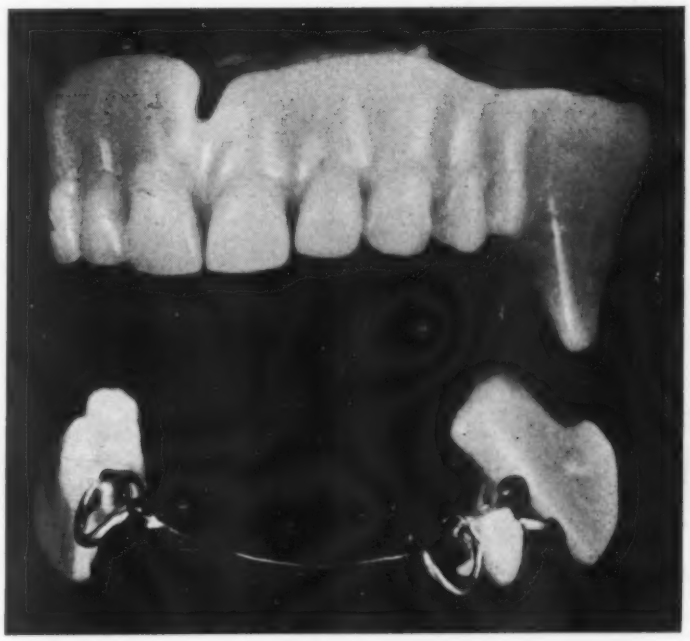


Fig. 7.—Dentures with flanges attached to guide mandibular fragment from midline into occlusion.

In cases where the insertion of a bone graft is to be postponed indefinitely or is contraindicated and yet there is only moderate deviation toward the midline, dentures with flanges with inclined planes on the affected side to guide the lower jaw into occlusion can be constructed (Fig. 7). These flanges are usually put on the side of the missing tissue in order not to interfere with mastication on the normal side. The length of the flanges will be determined by the vertical dimension of the patient's bite; the incline of the planes is such that upon contact and closure, the normal side of the mandible moves away from the midline into occlusion. The flanges may be on the buccal or on the lingual. When the remaining mandibular fragment has been markedly drawn toward the midline, a pseudotemporomandibular joint appliance, as designed by Dr. A. J.

Ackerman, may be constructed. This is essentially composed of a tube open at one end and with a slot running its entire length in one area; a bar with a rounded end that is of greater diameter than the shank, which fits into the tube, the other end of which is usually flattened and fits into a U-shaped device to form a hinge (Fig. 8). The hinge sometimes must be substituted by a smaller inferior tube, open end distad, so that the usually flattened end of the bar is also rounded. This would allow greater freedom of movement of the mandible where necessary. The superior tube, open end anteriad and the slot down, is attached to the upper denture or is soldered to crowns on maxillary molars on the side of the defect as far distad as possible. The U-shaped device is incorporated into the lower appliance and is united to the bar by means of a pin.

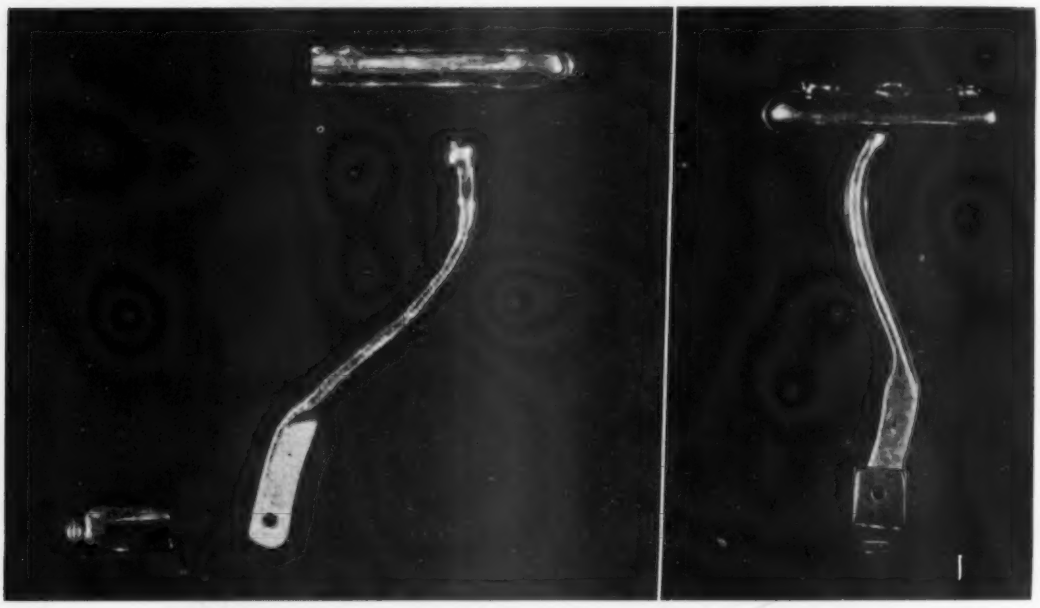


Fig. 8. Fig. 9.

Fig. 8.—Pseudotemporomandibular joint appliance—component parts. (Designed by Dr. A. J. Ackerman.)

Fig. 9.—Joint appliance assembled.

The bar connects the upper and lower appliances (Fig. 9). Added retention by means of special clasps may be necessary in the lower denture. The design may vary slightly, depending on the case, but the purpose of the completed appliance will be to eventually bring the remaining fragment away from the midline into occlusion with the opposing maxillary teeth. In completely edentulous cases, the joint appliance, modified to use an elastic band, is placed on the affected side and a spiral spring fitting into a groove in the upper and lower dentures is used on the normal side. These additions help stabilize the lower denture.

CORRECTIVE APPLIANCE FOR ANTERIOR SEGMENT OF JAW

In cases where the anterior segment of the mandible, for example in the region of the symphysis, is missing, the procedure is to construct an appliance (a cast or sectional or arch wire splint) that will prevent the two lateral segments

from collapsing toward the midline (Fig. 10). A cartilage or bone graft should then be inserted whenever possible. In no event should the collapsed segments be allowed to unite and heal. In an edentulous patient with a loss of this type, a satisfactory splint may be constructed as follows: An aluminum impression tray is trimmed to fit over the ridges. Holes for retention are drilled over the ridge area (Fig. 11). Softened compound is next added to the top and bottom

Fig. 10.

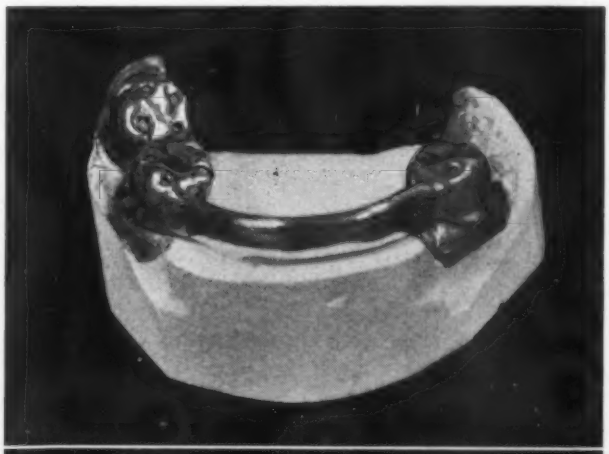


Fig. 11.

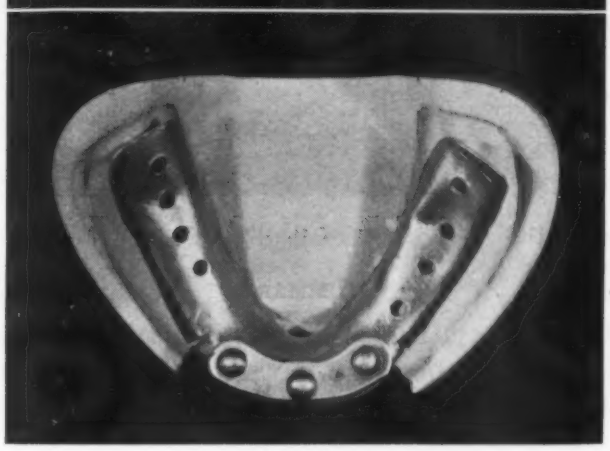


Fig. 10.—Cast sterling silver cap splint designed to prevent two lateral mandibular segments from collapsing toward the midline.

Fig. 11.—Tray splint used to prevent the collapse of lateral mandibular segments in edentulous patients.

of the tray, the combination positioned over the correctly aligned mandible, and the patient told to bite down into the compound; the latter is then cooled. The jaws are fixed with the aid of some form of head bandage. In cases where a graft is contraindicated, a fixed appliance, of which as much as possible is constructed before the operation, to hold the segments apart, is made with provisions for the insertion of a removable lip plumper (to facilitate oral hygiene). This can be constructed with or without teeth attached. Sometimes an anterior segment prosthesis is constructed for the purpose of acting as a scaffolding for plastic reconstruction of associated missing portions of the lower lip and chin (Fig. 12).

When an extensive portion of the horizontal ramus is missing, a chrome alloy appliance, in the form of the lost member, may be imbedded in the resulting cavity, its two free ends being attached to the cut ends of the ascending rami.[10] This procedure will tend to preserve the contour of the jaw and restore some degree of function of the mandible. If no appliance can be inserted and the contour of the chin has been lost, a hollow type artificial chin made of latex or similar substance can be constructed and attached in position on the face to restore its normal appearance.



Fig. 12.—Patient with portions of the lip and chin surgically removed

SUMMARY

When portions of the mandible are lost, a prosthesis should be constructed to restore occlusion and masticatory function and to improve appearance. The appliance may be a temporary one, being used as an adjunct to a bone graft, or may be a permanent one substituting for the lost tissue.

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Volume II

SUPPORTIVE THERAPY IN ORAL SURGERY

HERBERT J. BLOOM, D.D.S., M.S.*

ORAL surgery literature is abounding with detailed descriptions of manual operations, many of which have far-reaching systemic effects, with little regard for the general management of the patient undergoing the procedure. The oral surgeon has long since passed beyond the status of a skilled craftsman whose responsibility ceases with the tying of the last knot. The successful outcome of an oral surgical procedure or problem may directly depend upon the evaluation of the patient, the correction of associated disease, and the establishment of mental tranquility. Supportive therapy includes those measures specifically instituted to enhance the patient's security, augment his comfort, and promote healing with rapid return to health.

SEDATION

Surgery to the average person is an appalling experience associated with great mental trauma. The recognition and treatment of undue apprehension will make for a more rapid, uneventful convalescence. Probably the most widely used drug for prolonged sedation is phenobarbital. It is slow in action but long lasting. As it tends to be accumulative, untoward symptoms may be expected following its use over long periods of time. Its administration in doses of ½ grain at four-hour intervals will usually allay mental unrest.

Triple bromides, 5 to 10 grains in 1 to 3 ounces of water, given two to three times daily, will produce mental calm without hypnosis. Drugs having a pronounced hypnotic action may be required for the excessively nervous individual. In this regard the various members of the barbital family are to be recommended. Barbital, 7.5 grains, is slowly excreted and therefore long lasting; or the soluble barbiturates, Nembutal (Abbott) or Seconal (Lilly), in doses of 1½ grains are more profound in their action with earlier elimination. The latter two combined with aspirin, 5 to 10 grains, are effective in relieving mild discomfort. Sterile sodium phenobarbital, 2 grains, may be given subcutaneously to patients unable to take anything by mouth.

Other valuable time-honored hypnotics include: paraldehyde, 20 c.c. in water, as a retention enema; and chloral hydrate, 15 to 30 grains. The former

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may be objectionable because of its disagreeable odor, and the latter may occasionally produce a slight nausea in addition to being regarded as a respiratory and circulatory depressant.

Preoperative medication demands careful consideration, as it has been adequately demonstrated by experimental studies that the judicious use of preliminary medication will decrease morbidity and mortality during operative procedures. The reflex irritability of a patient is reduced by removing the factors of pain and fear, thus eliminating the second or excitement stage of general anesthesia. Sedation is equally as necessary with local anesthesia as with general anesthesia. Not only does it help relax the patient, thereby lightening the burden of the surgery, but the barbiturates counteract, by physiochemical means, much of the undesirable effects of many of the commonly used local anesthetic agents. It is a maxim that the full effect of the drug be reached when the induction of the anesthetic is begun, thereby merging normal sleep with surgical anesthesia. Should the maximum effect of the drug be attained when the patient is deeply narcotized, the combined effect may be sufficient to produce serious anesthetic complications.

The short-acting derivatives of barbituric acid, such as Seconal, are effective preoperative medicaments. They are usually administered at bedtime the night before surgery, and again forty-five minutes to one and one-half hours before the patient is taken to the operating room. The adult dose is 1½ to 3 grains. Children safely take ¼ to 1 grain, with the dose doubled for rectal administration. These barbiturates may be used with the popular combination of morphine sulfate ¼ grain and atropine sulfate ⅟₁₅₀ grain. The morphine and atropine are given forty-five minutes preoperatively with the barbiturate being given one hour earlier.

ANALGESIA

The relief of pain is one of the essential purposes in the practice of medicine and dentistry. Man has striven to gain this end from time immemorial. Prolonged intractable pain is exhausting and decreases tissue reserve necessary for recovery. The removal of pain promotes comfort and is a pillar in the supportive care of the patient. Relief of pain does not always necessitate the use of drugs, as discomfort can often be relieved by the manual correction of many factors. Decreasing pressure by the drainage of fluids, correction of a faulty dressing, or even change in bed position may add to the patient's comfort and increase the feeling of well-being.

Medication given to control pain should at all times be limited to the minimal dose necessary to achieve its purpose. Persons requiring long periods of analgesics, as, for example, in case of the inoperable carcinoma, may not obtain adequate relief at a time when it is most necessary because of early acquired tolerance for the drug. Also, it must be borne in mind that the pain-threshold-raising action of analgesics is reduced by pain. In other words, if pain precedes the administration or course of an analgesic, the effect of the agent is decreased and larger doses are required to bring about a desired result.

The most commonly used pain-depressing drugs are the analgesic-antipyretic group. They are used singly or in combination. Aspirin, 5 to 10 grains every two to three hours, is known to be effective against moderate pain. Phenacetin, 5 grains, and acetanilid, 3 grains, are equally acceptable, but their use for patients with heart disease is ill-advised. Empirin compound is a proprietary representing this group in combination. It is obtainable in tablet form combined with codeine phosphate, ¼ grain (#2), or ½ grain (#3), and relieves moderately severe pain in doses of 1 to 2 tablets every three hours if necessary.

Severe pain may necessitate the use of narcotics. Codeine, ½ to 1 grain, and morphine, ⅙ to ¼ grain, are potent analgesics. These are best administered hypodermically and at intervals not in excess of every three hours. Morphine, being a respiratory depressant, requires strict observation of the patient during its usage. Many individuals respond to morphine by an increase in nausea, in which event pantopon, ⅓ grain, may be substituted although it is less effective than morphine in combating pain. Dilaudid, 1/32 grain, is also less nauseating but has a distinct respiratory depressant action. Recently Demerol (Winthrop) has become popular as an analgesic. It is administered in 100 mg. doses intramuscularly or orally. Its antispasmodic action makes it particularly applicable for pain associated with smooth muscle spasm. Its position in the analgesic scale is between codeine and morphine.

FLUIDS

The properly hydrated patient is a better surgical risk with less tendency toward complication and delayed recovery than one dehydrated or improperly hydrated. The administration of fluids requires a knowledge of fluid and electrolyte balance with due regard for the amount and type of fluid indicated. Normally the kidneys excrete approximately 1,000 to 1,500 c.c. of fluid containing 35 to 50 grams of urinary solids daily. In addition, about 300 c.c. of fluid is lost in the expired air, 200 c.c. in the feces, and up to 1,500 c.c. from the cutaneous surfaces. As fluid intake should equal output, 3,000 to 3,500 c.c. are the minimal daily requirement to maintain water balance. Although water represents approximately 70 per cent of the body weight, a loss of fluid equivalent to as little as 6 per cent of the body weight will produce signs of severe dehydration and intoxication. On the other hand, the ill-advised forcing of fluids may result in edema, which in the presence of cardiac or renal disease may overtax the diminished reserve of these organs to the point of fatal consequence.

The type of fluid is as important as the amount. For example, the person who consumes copious amounts of water may manifest signs indicative of dehydration. The excess fluid eliminated by the kidneys carries with it sodium chloride. The removal of salt depletes tissue storage, and tissue fluid must necessarily be withdrawn to maintain isotonicity, and dehydration results. Likewise, if excess saline solution is administered, the retention of salt requires fluid to produce osmotic balance, and edema results.

Although there are early clinical signs of fluid need (dryness of the lips and tongue, increased pulse and respiration rate, and anorexia), the definite

clinical picture of dehydration occurs late in the process. Other than the knowledge that fluid intake has been inadequate, the need for replacement is readily determined by laboratory studies. A urinary output below 600 c.c. daily of high specific gravity is usually indicative of insufficient fluid intake. The nitrogenous content of the blood increases, and the urea nitrogen (normal 12 to 15 mg.) and the nonprotein nitrogen (normal 25 to 35 mg.) will show a rise. These findings, however, may be simulated in renal disease with sufficient fluid intake. The blood shows other characteristic changes. As the fluid content of the blood decreases, the concentration of blood cells and blood plasma increases, with a rise above probable normal values in hemoglobin and red cell count.

If dehydration occurs as a result of persistent vomiting, chlorides are lost. The excess retained sodium ions combine with the bicarbonate of the blood and tissue fluid to form sodium bicarbonate producing alkalosis. The blood plasma chlorides are below normal (525 mg. per cent), the carbon dioxide combining power of the blood is increased (normal 50 to 65 volumes per cent), and the urine is basic to litmus. Decreased urinary output predisposes the retention of acid metabolites which are notably formed in diabetes mellitus and malnutrition. These retained acids combine with sodium, displacing bicarbonate which is excreted through the lungs in the form of carbon dioxide. Respiration is necessarily more rapid, the carbon dioxide combining power of the blood decreases, the urine is acid to litmus, and acetone is found in the specimen. The patient is said to be in acidosis.

The need for fluid having been determined there are certain basic principles of fluid therapy. The total intake must be sufficient to maintain a minimal urinary output of 1,000 c.c. for a twenty-four-hour period. This will usually require a daily minimum of 3,000 c.c. of fluid. The type of fluid varies with the etiology of dehydration; while normal salt solution will correct acidosis or alkalosis, it must never be given to the degree that edema is produced. Acidosis in a dehydrated patient is more readily corrected by the additional use of 10 per cent dextrose solution. While dextrose solution supplies not only needed fluids but caloric requirement as well, its extreme use must be guarded, as salts may be removed with the excretion of the fluid. Five per cent dextrose solution, however, is so nearly isotonic that it may be given in relatively large amounts with a fair degree of safety.

If fluid requirement cannot be maintained by oral route, intravenous infusion is the parenteral method of choice. Usually 5 per cent dextrose, normal saline, or a combination of these is used. The solution may be delivered in quantities of 500 to 1,000 c.c. at intervals, or the continuous intravenous drip method may be used. Nutritional deficiencies may be corrected by the addition of nutritional elements to the fluid.

NUTRITION

The ingestion of an adequate amount of the essential food elements necessary for the return to and maintenance of a state of health is a prime factor in the support of the surgical patient. It is a professional duty to understand

the relationship between nutritional deficiencies and tissue recovery. A detailed discussion of the necessary elements of a well-balanced and assimilable diet is far beyond the confines of this paper. For the sake of completeness in reviewing supportive therapy, a few remarks concerning fundamentals are in order.

Nutritional deficiencies are of frequent occurrence in the oral surgical patient—usually because of existing obstacles in the taking of all forms of food by mouth. Borderline or subclinical deficiencies which are intensified by a surgical ordeal or short starvation period are not uncommon.

The intake of carbohydrate is a requisite for the debilitated patient as it will help meet caloric requirements and uphold the glycogen stores of the liver. An average adult weighing 150 pounds has a minimum requirement of 1,600 calories daily. An elevation of body temperature will increase the caloric needs. In the event of depletion of the carbohydrate reserve, the body fat deposits are called upon to meet the energy demands. Some carbohydrate is essential for the complete metabolism of fat, otherwise nonvolatile metabolic acids are produced and a state of acidosis results. It is sound practice to force a high carbohydrate intake for several days preoperatively, or to give 5 to 10 per cent dextrose intravenously immediately before operation. This furnishes a reserve for the day of surgery and for the several postoperative days when food intake is likely to be low.

Protein adequacy is a significant factor in healing and preventing wound complications. In marked protein deficiency there is loss of plasma proteins with resultant decrease in plasma fluid to keep blood protein concentration and osmotic pressure with tissue fluids in a state of balance. Infusion of the usual fluids (dextrose or saline) dilutes the plasma proteins and accentuates the clinical picture of hypoproteinemia. The normal level of plasma protein concentration (7.0 grams per cent) must be maintained, and if deficiency is suspected a laboratory determination is indicated. The use of a high-protein diet is the simplest method of restoring protein requirements. Should this method be impractical, whole compatible blood or plasma transfusions will correct the condition.

Nutrition is a positive force in health and the vitamins are a positive force in nutrition. Their inclusion in dietary measures helps compose nutritional adequacy, and while they are not a panacea for all the ills of man, their importance in helping a patient attain a higher level of health cannot be overstressed. To obtain optimal nutrition for a patient on restricted foods it is imperative that the diet be supplemented with these essential factors. While the exact number of vitamins is still unknown, ten have been studied with a fair degree of thoroughness and the effects of their inadequate intake have been well established. Certain facts concerning a few of these may be of interest here.

Vitamin A is essential for the normal functioning of epithelial surfaces. It is known to increase the resistance of the body to infections making their entrance through the respiratory and urinary tracts. In deficiency states it is responsible for the production of poor appetite, gastrointestinal disturbances, and a condition of the eye known as xerophthalmia. The adult daily minimum requirement is 6,000 International Units.

Vitamin B is known to be a complex mixture of many vitamins. In this group are included the following:

Thiamine, a requisite in the metabolism of carbohydrates, and essential for the normal condition and functioning of nerve tissue. Deficiencies produce generalized weakness, anorexia, gastrointestinal and cardiovascular disturbances, neuritis, and in the surgical patient will delay healing. The adult daily minimum requirement is 333 International Units (1 milligram).

Riboflavin is essential for proper functioning of the gastrointestinal tract, maintenance of a healthy skin, and normal functioning of the visual apparatus. Deficiencies are responsible for "commissural cheilosis" and keratitis. The adult daily minimum requirement is 2,000 micrograms.

Niacin has been claimed as the pellagra-preventive vitamin. Its deficiency produces skin eruptions, inflammations of the alimentary tract, and derangement of the central nervous system. It has been suggested that ulceromembranous stomatitis is a subclinical pellagral manifestation that responds to therapeutic doses of niacin. The adult daily requirement is 15 to 20 milligrams.

Other components of vitamin B, vitamin B_6 and pantothenic acid, are, to date, of lesser importance in supportive dietary measures.

Vitamin C (ascorbic acid) is responsible for the formation of intercellular cement substance. Its importance in wound healing has been unquestionably determined. Advanced deficiencies result in a condition known as scurvy, characterized by capillary hemorrhages, swollen joints, edematous gums, loose teeth, and anemia. The adult daily minimum requirement is 30 milligrams.

Vitamin D is of importance in conditions involving the healing of bones. It regulates the metabolism of calcium and phosphorus and is essential to bone formation. The deficiency of this vitamin produces poor assimilation of ingested calcium and phosphorus and, therefore, poor bone formation. The therapeutic administration of vitamin D requires that calcium and phosphorus in adequate amounts also be provided. The adult daily minimum requirement is 400 International Units.

In general, it may be said that the routine administration of multivitamin preparations to the oral surgical patient, even in the absence of clinical evidence of vitamin deficiency disease, is a sound supportive measure. The choice of such a drug depends upon the specified potency of its vitamin content and upon the reputation of the pharmaceutical house engaged in its manufacture. Many such suitable preparations are available to the profession.

ELIMINATION

The normal motility of the digestive tract is usually decreased in the person confined to bed. Further, lack of bulk in the diet predisposes the patient to constipation. While this may not seriously influence the course of convalescence, it may produce abdominal distress and generalized discomfort. Often satisfactory bowel function can be obtained by including in the diet, if possible, adequate amounts of the laxative foods (oatmeal, prunes, whole-wheat bread, honey, and syrup). If more potent peristaltic stimulants are required, enemas are satisfactory. Small enemas (1 pint of warm tap water or soapsuds) are desirable. Equal parts of water and glycerin (about 4 ounces), or 3 to 6 ounces of olive or mineral oil, as a retention enema, will act as a slight rectal stimulant and produce the desired results.

Liquid petrolatum, 1 ounce at bedtime, is usually satisfactory in relieving ordinary constipation. Its continued use is to be discouraged as it tends to retard absorption of foods by forming a coating over the food particles and gastric mucosa. Any artificial peristaltic stimulant over a long period will decrease the normal defecatory reflex.

CORRECTION OF ASSOCIATED DISEASE

It is understood that the treatment of an existing intercurrent systemic disease is outside the realm of the oral surgeon and demands consultation with the internist. A close cooperation between the dental and medical professions acts for the welfare of the patient. Nevertheless, it behooves the oral surgeon to familiarize himself with the more common systemic complicating factors as their early recognition may significantly alter the course of the primary disease.

Anemias are not an uncommon occurrence in the patient under oral surgical care. Most often they are of the chronic secondary form and may accompany any disease condition in the body. The oral surgeon frequently sees anemias resulting from blood loss and infection. It is legion that anemias occur in many middle-aged women—probably from menorrhagia. The anemia of blood loss is of the normocytic type and is manifested by a decrease in the number of red blood cells with a corresponding quantitative decrease in hemoglobin percentage. Other of the so-called secondary anemias are of the microcytic hypochromic type with a decrease in hemoglobin out of proportion to the decrease in the number of red blood cells.

Regardless of the etiology of the anemia it must be corrected to assure tissue recovery. Because the anemic patient has diminished oxygen-carrying capacity of the blood and is highly susceptible to shock, surgical procedures must be delayed until the condition has been corrected. Iron, being essential for the metabolism of hemoglobin, is the drug used for the treatment of this condition. Ferrous sulfate, 15 grains daily in divided doses, is a most efficient preparation in this regard. Ferrous gluconate may be used instead for those patients having an intolerance to oral iron.

Hemorrhagic tendencies occasionally present a problem, and persons giving a definite history of prolonged bleeding following relatively minor surgical trauma demand careful investigation and definite control before instituting surgical therapy. There are a few noteworthy systemic diseases having this manifestation.

Hemophilia, a qualitative disease of the blood platelets, produces delayed clotting time although bleeding time is within normal range.

Jaundiced states indicate that bile is being excluded from the intestinal tract. Vitamin K, an antihemorrhagic factor necessary for the production of prothrombin, requires bile for its absorption from the intestinal tract. Its deficiency occurs when bile is lacking and decreased synthesis of prothrombin results, producing a prolonged clotting time. If Vitamin K is given as a therapeutic measure it should be given in conjunction with bile salts.

Thrombocytopenic purpura is a quantitative disease of the blood platelets. The bleeding time is prolonged; the clotting time is within normal range.

Hypoparathyroidism may result in a decrease in blood calcium ions. As this element is essential in the formation of a blood clot, a prolonged clotting time results.

Scurvy is a disease of vitamin C deficiency and is manifested by increased capillary fragility and bleeding tendency. The constituents of the blood are unchanged.

Of the hemorrhagic diseases, hemophilia and thrombocytopenic purpura are the most difficult to control, and the preparation of these patients for operation requires long, arduous, and careful study.

Many other commonly occurring, coincidental complicating systemic diseases require judicious treatment by the internist to decrease operative risk or support recovery. Certainly, the uncontrolled diabetic requires the very closest cooperation between surgeon and internist to carry the patient safely through surgical therapy and tissue healing. These persons are notoriously prone to infection and recovery is likely to be complicated.

Renal diseases result in disturbed fluid and electrolytic balance and must be adequately dealt with.

Cardiac disease although not usually altering the progress of recovery must be closely watched to insure the patient's safety during a surgical ordeal.

Further useful procedures in the restoration and procurement of comfort for the exhausted patient include: conservation of body heat, proper position of the patient, and rest. Debility predisposes the body to lessened resistance which may result from rapid decrease in body temperature. Body heat should be maintained by warmed, light blankets and by artificially heated atmosphere, rather than the exclusion of fresh cold air.

The position of the patient does much to augment his comfort and may be highly important as a safety measure. Most patients confined to bed for long periods and immediate postoperative cases do best in Fowler's or semi-Fowler's position. This is particularly true when respiratory difficulty is a factor to be contended with, as respiratory movements have less interference in this position. Frequent change in position will decrease the possibility of decubitus, joint disturbances, and sometimes pulmonary complication. It may be necessary to alter the position of a patient to provide better drainage from an abscess.

Rest is a fundamental requisite in supportive measures. It includes much of the afore-mentioned material: sedation, control of pain, and comfortable position. Noise and strong light must be excluded. Above all, it includes such mental comfort as may come from reassurance and a confident, cheerful, sympathetic attitude on the part of the surgeon.

SUMMARY

The adequate therapy revolving around the care of the oral surgical patient includes more than the skillful carrying out of a technical procedure. It includes specific systemic measures designated to increase the rapid return to a

state of health. Among these measures the oral surgeon must consider adequate sedation and control of pain. He must be certain that his patient is properly hydrated and that nutrition is adequate for tissue healing.

Concomitant systemic diseases which are likely to retard the patient's progress must be controlled. Close cooperation between oral surgeon and internist makes smoother, more rapid convalescence with enhanced security for the patient.

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ESSENTIALS FOR SUCCESSFUL ROOT RESECTION

RALPH F. SOMMER, D.D.S., M.S.*

THE surgical removal of periapical pathology, first advocated by Farrar[1] in 1875 and later given greater emphasis by Rhein[2] in 1890, has come to be accepted for many years as good operative procedure. In view of the close similarity of the various techniques that have been published from time to time, it would seem that little if anything of a contributing nature could be added to the present store of knowledge of the operation. Aside from some differences of opinion as to whether resection had better be done prior to or after the treatment and filling of the canal, one may draw the conclusion that, whichever way is followed, good results are very apt to occur provided all pathology has been removed and the tooth rendered tissue tolerant. A brief review of some of the techniques advocated in the past will serve as a basis for the adoption of present-day concepts.

In 1911, Dr. Thomas B. Hartzell[3] of Minneapolis advocated the use of a trephine for establishing a large opening over the area of pathology, and that drainage should be maintained until the wound had healed. By 1915 Dr. Immanuel Ottesen, Dean of the School of Dentistry in Oslo, Norway,[4] demonstrated the open-view method for root resection at the Dental School, University of Minnesota. An incision was made, the soft tissues retracted, the alveolar bone over the apex removed with a chisel, the end of the root amputated and the area curetted, an amalgam filling inserted in the apex, an antiseptic placed in the wound and the tissues sutured at the line of incision. No mention was made about the status of the canal prior to the operation, nor the causative factors responsible for the pathology which prompted it.

Following closely the technique advocated by Dr. Ottesen, M. H. Garvin,[5] of Winnepeg, Manitoba, reports having performed resections on 1,179 teeth from 862 patients. Instead of limiting himself to anterior teeth, Garvin included the premolars and first molars in many instances. The extraction of teeth that he had resected twenty-two years previously bears evidence to the fact that his technique must have been quite successful. Unfortunately, with the approach of the focal infection "era" in 1920, Garvin, like many others, heeded the advice of uninformed believers in the then novel theory and for a time discontinued the practice of root resection, in spite of the excellent results he had obtained, only to revive the practice and continue it up until the present time.

Although advocates of the focal infection theory, at the time, gained sufficient support to discourage the conservation of pulpless teeth throughout this country, the University of Michigan Dental School gave encouragement

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to such men as U. G. Rickert and Chalmers J. Lyons to keep the subject alive. Fully aware that pulpless teeth showed evidence of periapical pathology, which in many instances was brought about through the invasion of microorganisms, Dr. Rickert, bacteriologically trained and therapeutically minded, pioneered in the direct application of bacteriologic methods in the more rational control of infected pulpless teeth. Although oral surgeons generally were prone to accept the validity of the focal infection theory to justify the removal of questionable teeth, the conservative attitude adopted by Chalmers J. Lyons in his support of root resection again helped to stimulate renewed interest in this operation.

SIMPLIFIED OPERATIVE PROCEDURES THAT HAVE PROVED THEMSELVES SUCCESSFUL

During the past fifteen years, several thousand root resections have been performed in the postgraduate clinics of the Dental School at the University of Michigan, on patients ranging in age from 10 to 50 years. The routine procedure has been as follows:

1. After assignment to the postgraduate student, carefully timed and standardized radiographs are obtained.

2. Inasmuch as root resection involves bone and soft tissue surgery, requiring an anesthetic, it is imperative that an adequate medical history of the patient be taken. Dentists enrolled as postgraduate students are usually insufficiently trained in physical diagnosis to obtain a reliable medical history, and therefore are given special instruction by physicians at the University Hospital.

In Fig. 1 it will be noted that the left half of the history form pertains to the clinical history of the teeth to be operated upon. The right half pertains to the general health of the patient. Information obtained in the history not only may be invaluable in planning the treatment of the patient, but in some instances may definitely contraindicate the surgery that had been planned.

The reverse side of the history sheet constitutes a work chart, shown in Fig. 2.

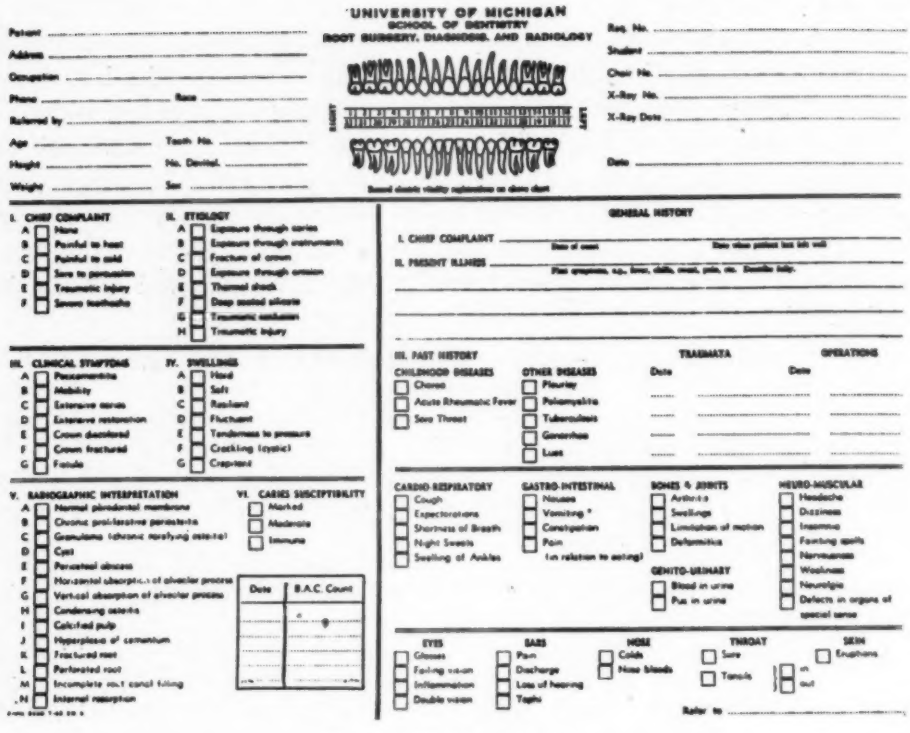
The length of the tooth, taken by means of delicate exploration with marked instruments, is recorded on the scale marked *6. Computed Tooth Length.*

The importance of maintaining proper length control throughout the course of the preliminary root canal therapy cannot be overemphasized. Acute exacerbations are brought on through overzealous instrumentation causing inoculation of the periapical tissues with poisonous canal contents. The careless use of root canal files may not only result in inexcusable breaking of the file in the canal, but also cause root perforations and painful injury to the periapical tissues. The correct length of the canal is obtained by carefully feeling for the constriction at the dentocemental junction with a No. 1 Kerr file fitted with a rubber marker coinciding exactly with the cutting edge of the tooth. All subsequent instruments should be marked accordingly and carried to the same length.

The great majority of teeth to be resected are previously treated until negative cultures are obtained from the canal. The drugs used and the bacteriologic findings following each treatment are recorded on the section of the chart marked *8.* Further mention of this will be made later. The operative procedure for each resection is accurately recorded in column 7. All pathologic tissue removed from the periapical region is biopsied, the findings to be reported by the Dental School pathologist, Dr. Kerr.

Since all preliminary root canal work is carried out under the strictest bacteriologic asepsis, the following armamentarium is essential:

An aseptic field is obtained by means of a simplified rubber dam technique.



UNIVERSITY OF MICHIGAN
SCHOOL OF DENTISTRY
ROOT SURGERY, DIAGNOSIS, AND RADIOLOGY

Patient
Address
Occupation
Phone — Race
Referred by
Age — Teeth No.
Height — No. Dentist
Weight — Sex

Req. No.
Student
Chair No.
X-Ray No.
X-Ray Date
Date

I. CHIEF COMPLAINT
A None
B Painful to heat
C Painful to cold
D Sore to percussion
E Traumatic injury
F Severe toothache

II. ETIOLOGY
A Exposure through caries
B Exposure through instruments
C Fracture of crown
D Exposure through erosion
E Thermal shock
F Deep seated silicate
G Traumatic occlusion
H Traumatic injury

III. CLINICAL SYMPTOMS
A Percussionitis
B Mobility
C Extensive caries
D Extensive restoration
E Crown discolored
F Crown fractured
G Fistula

IV. SWELLINGS
A Hard
B Soft
C Resilient
D Fluctuant
E Tenderness to pressure
F Crackling (cystic)
G Crepitant

V. RADIOGRAPHIC INTERPRETATION
A Normal periodontal membrane
B Chronic proliferative periodontitis
C Granuloma (chronic apical periodontitis)
D Cyst
E Periapical abscess
F Horizontal absorption of alveolar process
G Vertical absorption of alveolar process
H Condensing osteitis
I Calcified pulp
J Hyperplasia of cementum
K Fractured root
L Perforated root
M Incomplete root canal filling
N Internal resorption

VI. CARIES SUSCEPTIBILITY
Marked
Moderate
Immune

Date	B.A.C. Count

GENERAL HISTORY

I. CHIEF COMPLAINT — Date of onset — Date when patient last felt well
II. PRESENT ILLNESS — Give symptoms, e.g., fever, chills, sweat, pain, etc. Describe fully.

III. PAST HISTORY

CHILDHOOD DISEASES
Chorea
Acute Rheumatic Fever
Sore Throat

OTHER DISEASES
Pleurisy
Poliomyelitis
Tuberculosis
Gonorrhea
Lues

TRAUMATA — Date
OPERATIONS — Date

CARDIO-RESPIRATORY
Cough
Expectorations
Shortness of Breath
Night Sweats
Swelling of Ankles

GASTRO-INTESTINAL
Nausea
Vomiting *
Constipation
Pain
(in relation to eating)

BONES & JOINTS
Arthritis
Swellings
Limitation of motion
Deformities

GENITO-URINARY
Blood in urine
Pus in urine

NEURO-MUSCULAR
Headache
Dizziness
Insomnia
Fainting spells
Nervousness
Weakness
Neuralgia
Defects in organs of special sense

EYES
Glasses
Failing vision
Inflammation
Double vision

EARS
Pain
Discharge
Loss of hearing
Tophi

NOSE
Colds
Nose bleeds

THROAT
Sore
Tonsils

SKIN
Eruptions

Refer to

Fig. 1.—History chart.

TREATMENT CHART

Date started ..... 19....
Date completed ..... 19....

1. CANAL MORPHOLOGY
A Anatomically normal
B Coronal calcification
C Constricted
D Foramen open
E Foramen constricted
F Previously filled
G Metal post

A B C D E Pulpotomy — Vital / Non-vital Fixation

2. RESTORATION CONTEMPLATED
A Silicate
B Amalgam
C Inlay Type
D Bridge abutment
E Clasp attachment
F Davis crown
G Jacket crown
H Richmond crown

Technic
Material used

3. INSTRUMENTATION

A	Occlusal opening	A B C D E
B	Canal not located	A B C D E
C	Apex not reached	A B C D E
D	Apex perforated	A B C D E
E	Side perforated	A B C D E
F	Shoulder	A B C D E
G	Finished canal wall	A B C D E

4. FINAL FILLING

A	Condensation	A B C D E
B	Length control	A B C D E

5. RADIOGRAPHIC TECHNIC
A B C D E Angulation (vertical)
A B C D E Angulation (horizontal)
A B C D E Exposure under over
A B C D E Processing Dev. Fix Wash
A B C D E Mounting

Comments

6. COMPUTED TOOTH LENGTH

A	Single canal	
B	M.B. canal	
C	D.B. canal	
D	M.L. canal	
E	Lingual	
F	Buccal	

Transfer marked file length to above scale.

7. ROOT RESECTION — Operated by — Assisted by

Tooth resected	A	
Incision	B	Dr. — Dr.
Conduct of patient	C	Anesthetic used
Type of incision	D	
Periapical findings	E	
Operative procedure	F	
Biopsy	G	No.
Prognosis	H	

8. THERAPEUTICS

Date	Drug

BACTERIOLOGY

Date	Culture Report	

Grade on radiographic dx. A B C D E
Grade on case management A B C D E
Grade on therapeutics A B C D E
Grade on bacteriology A B C D E

Grade on completed case A B C D E

Fig. 2.—Treatment chart.

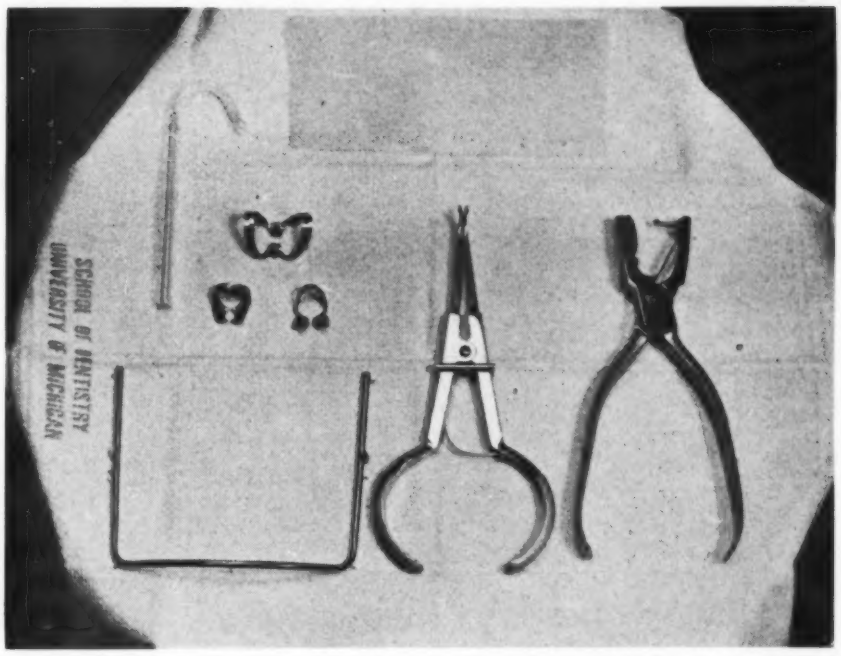


Fig. 3.—Tray set up for placement of rubber dam.



Fig. 4.—Punch hole 1 inch from upper border of rubber. Hold dam in place with thumb and middle finger, using index finger to center hole over tooth.

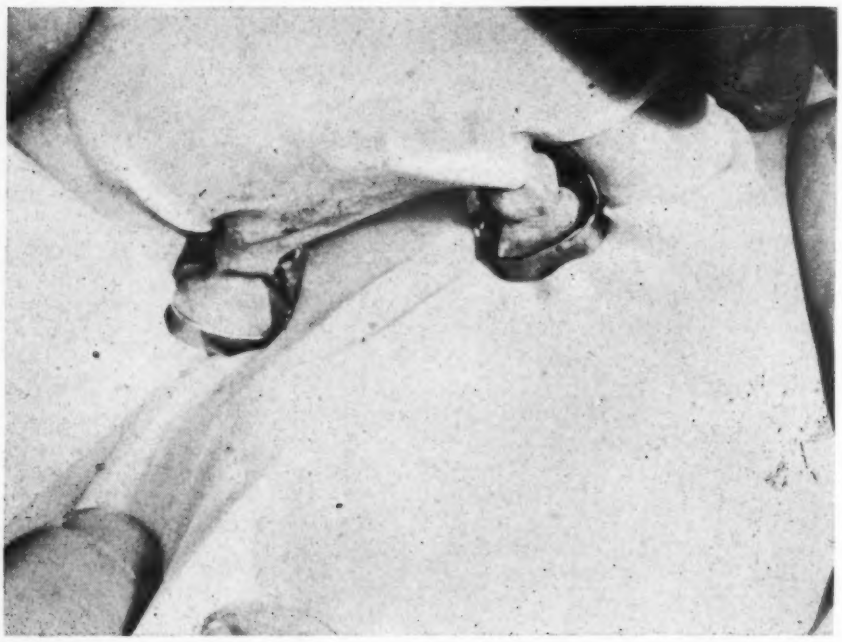


Fig. 5.—Carry S. S. W. clamp No. 27 over rubber to clasp premolars on both sides.

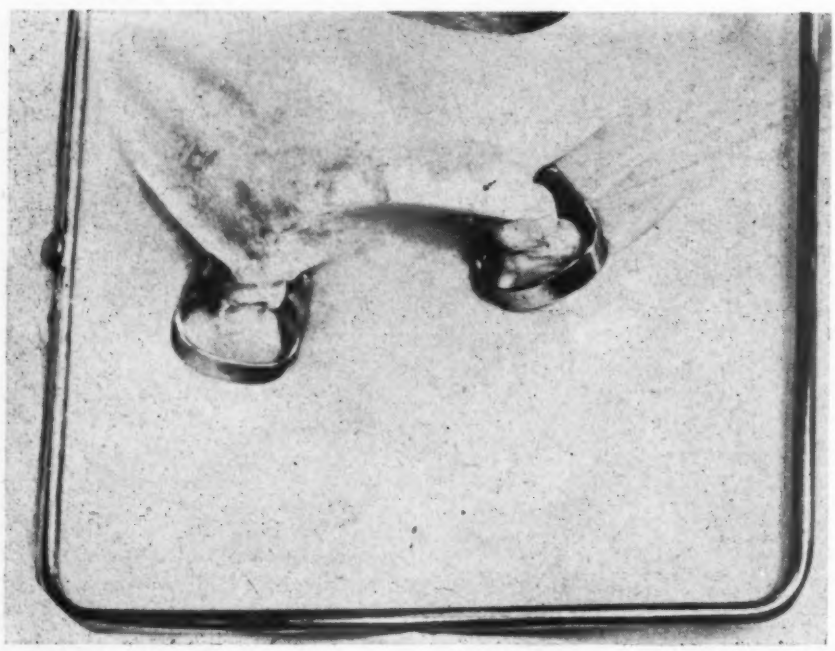


Fig. 6.—Put Young rubber dam frame into place, so as to eliminate as many wrinkles as possible.

The cumbersome method of painful ligatures, head halters, and weights has given way to a three-clamp combination and the Young rubber dam frame, making it possible to place the dam within a matter of fifteen or twenty seconds.

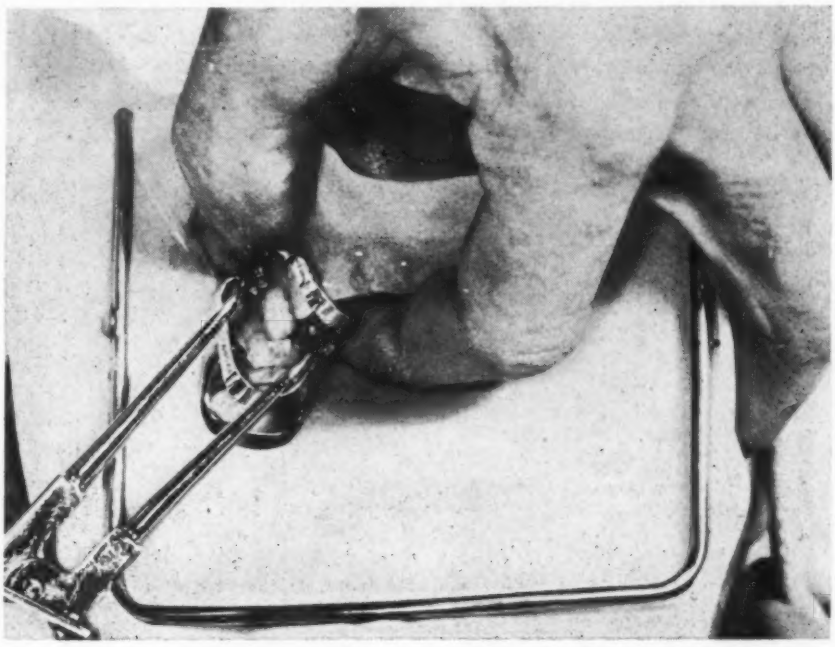


Fig. 7.—Isolate tooth to be worked upon with Ivory No. 9 double wing clamp.

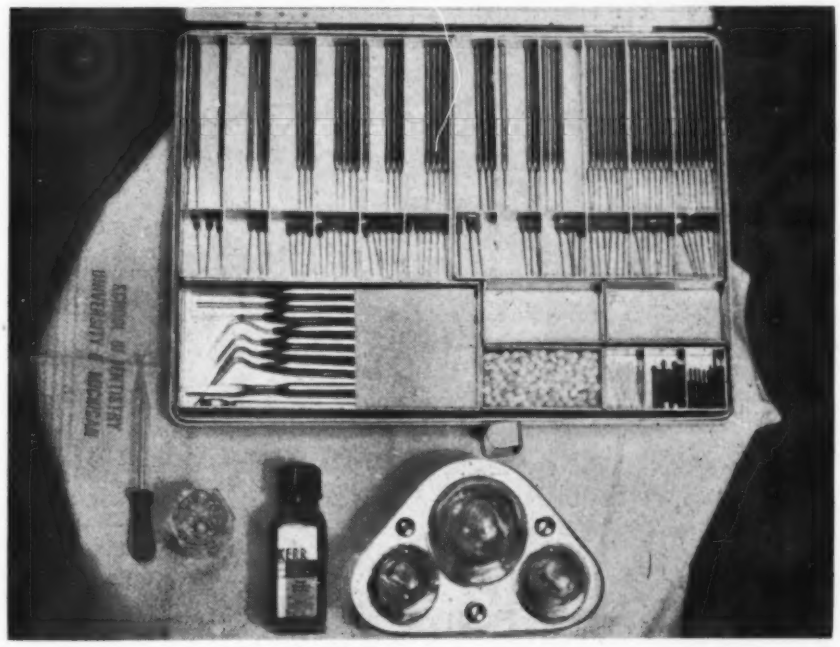


Fig. 8.

The area enclosed by the Young frame is then painted with tincture of Mercresin. Sterile instruments, paper points, and cotton pellets are kept in a Kerr aluminum case as shown in Fig. 8.

The entire case is sterilized in a dry heat oven at 160° C. or 320° F. At the time of use, root canal files are removed from sterile case with a flamed pair of cotton pliers and placed into the Kerr three-part well containing Metaphen germicidal solution. All accessory hand instruments, such as mouth mirrors, explorers, plastics, etc., are kept in a Bard-Parker sterilizer containing the above solution. While in use, hand instruments are placed in the folds of a sterile towel on the bracket table, so as to avoid air contamination.

Prepared culture media, ready for use, comes in hermetically sealed glass tubes with a separate sterile glass container for the sterile cotton plug.* (Fig. 9.)



Fig. 9.—When ready for inoculation, the cellophane seal is broken, the cap removed from the tube, the orifice flamed, and the sterile cotton plug inserted. The tube is now ready for use.

PRELIMINARY ROOT CANAL THERAPY ESSENTIAL FOR SUCCESSFUL ROOT RESECTION

The following drugs used in the preliminary root canal therapy are essential:

Alcohol
Phenol
Formocresol
Iodine lotion
Eugenol
Iodized phenol
Tri-Chlor or Zonite
Tincture of Mercresin
Bicarbonate of soda
Zylol or eucalyptol
Camphorated para-mono-chloro-phenol
28 per cent ammonium hydroxide
Silver nitrate crystals
Compound tincture of benzoin

*Made by the Difco Laboratories of Detroit.

The erroneous belief that root resection alone is sufficient to eliminate periapical pathology permanently is far from correct. Surgical measures alone, regardless of how skillfully performed, will not restore the periapical tissue to a normal physiologic status. Every source of irritation must be eliminated from the canal and prevented from returning to the periapical tissue by complete

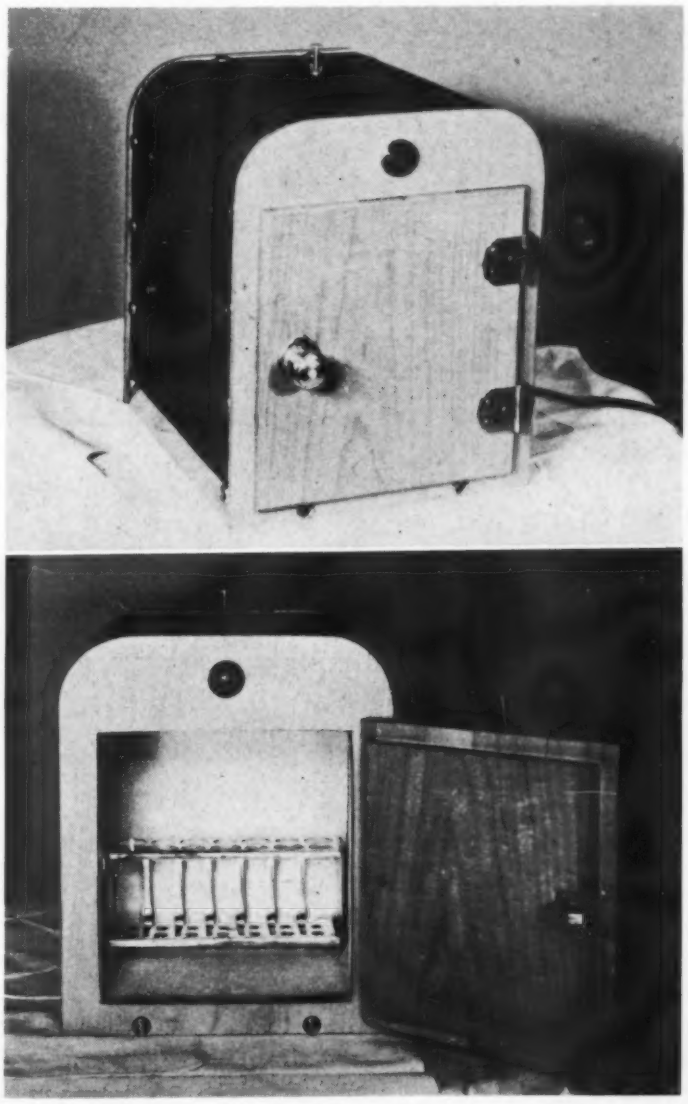


Fig. 10.—A suitable incubator for growing bacteria taken from the canals is essential. The incubator shown in Fig. 10, designed for use in the dental office, is so constructed as to maintain an even constant temperature of 37½° C.

hermetical sealing of a sterile canal with agents of known tissue tolerance. To illustrate this point, Fig. 12 shows a resected right lateral incisor that had been completed in the clinic. During the summer vacation the patient had returned to her home, only to have the opposite left lateral incisor develop an acute periapical abscess. Her family dentist recommended immediate resection to

clear up the abscess. The resulting destruction of all the supporting bone, not only involving the lateral incisor, but the cuspid as well, bears evidence to the fact that the resection did not solve the difficulty. The dentist neglected to treat and fill the canal. (Fig. 12.)

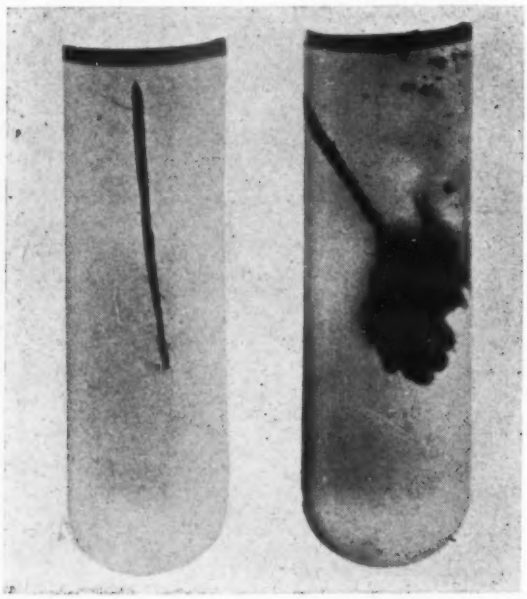
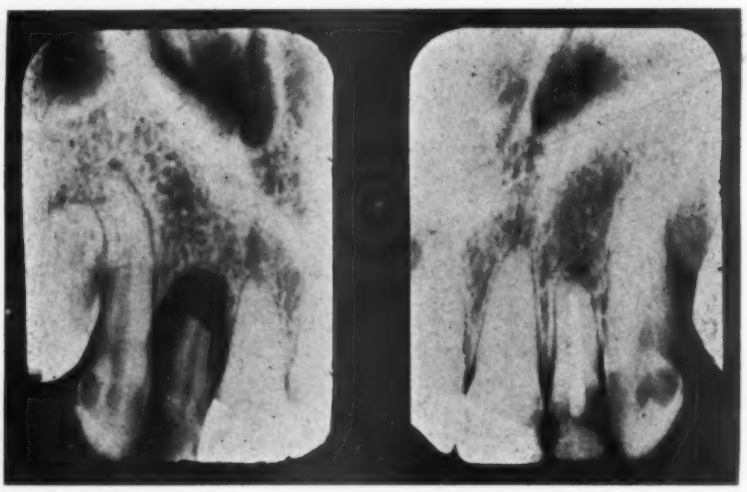


Fig. 11.—This shows a typical culture taken on a sterile paper point from an infected canal, and incubated for forty-eight hours. Note the luxurious growth at the tip of the paper point where it came into contact with live bacteria. Plain point shows negative culture following satisfactory treatment.



Left Fig. 12. Right

Since root resection is indicated only in teeth presenting evidence of periapical pathology, it is essential that an accurate diagnosis be made before any operation is contemplated. Careful vitality tests should be made to substantiate the radiographic findings, regardless of how obvious the latter may appear.

The general tendency to regard every radiolucent area at the apex of a tooth as indicative of pathologic tissue is incorrect (Fig. 13).

In Fig. 13 the lower five anterior teeth show radiolucent areas suggestive of granulomas. All teeth tested normal to ice and to the "Burton Vitalometer." This condition, although not too common, is brought on through traumatic occlusion, by either the opposing natural teeth or some restorative appliance. Relieving the bite will cause the radiolucent areas to disappear. This condition is known as bone hypoplasia. Treatment and subsequent resection are contraindicated.

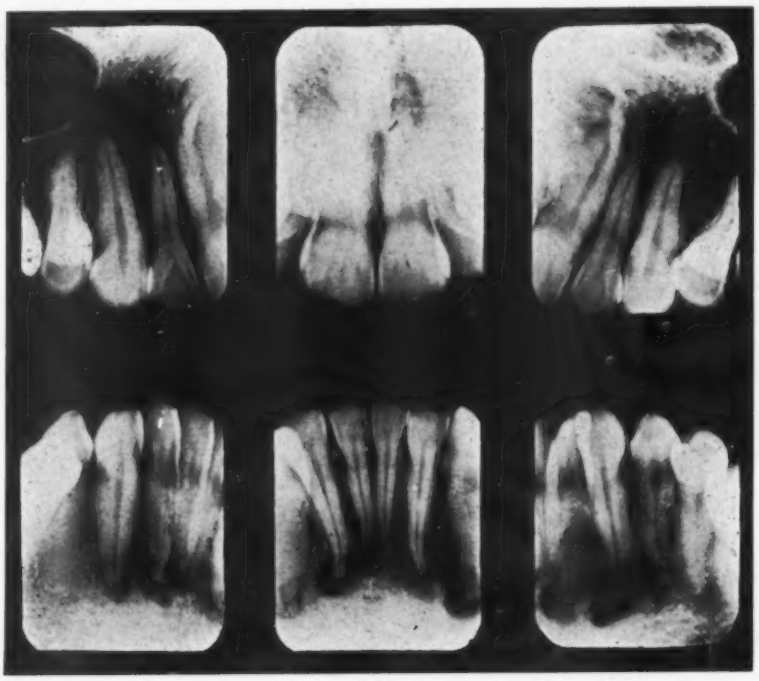


Fig. 13.

Pathologic findings from many resected areas have shown the periapical tissues to consist of pyogenic granulation tissue, in most instances representing a form of osteomyelitis. In order to avoid the hazard of reinfection following resection, it is advisable to eliminate or attenuate, insofar as possible, the bacteria present, by previous medication. Two or three treatments at forty-eight-hour intervals with camphorated para-mono-chloro-phenol will produce negative cultures. The use of hot sulfonamides and penicillin has been suggested in an effort to minimize the number of treatments.

If, at the time of treatment, all cultures are negative at the outset, in spite of the presence of a radiolucent area, root resection may not be necessary.

In Fig. 14 an extensive area of radiolucency is noted between two lower central incisors following a blow in a 16-year-old patient. After three successive cultures (Fig. 15), all of which were negative, the canals were filled with silver points. Complete return to normal bone architecture followed without resection having been necessary.

The mere fact that negative cultures have been obtained from the canal does not signify that all the dentinal tubules are equally sterile. In order to render

the entire body of the dentine free from foreign matter, thus making the entire body of the tooth tissue tolerant, a powerful solution of dehydrated phenol sulfonic acid is used in the canal for a few minutes just prior to the filling of the canal.

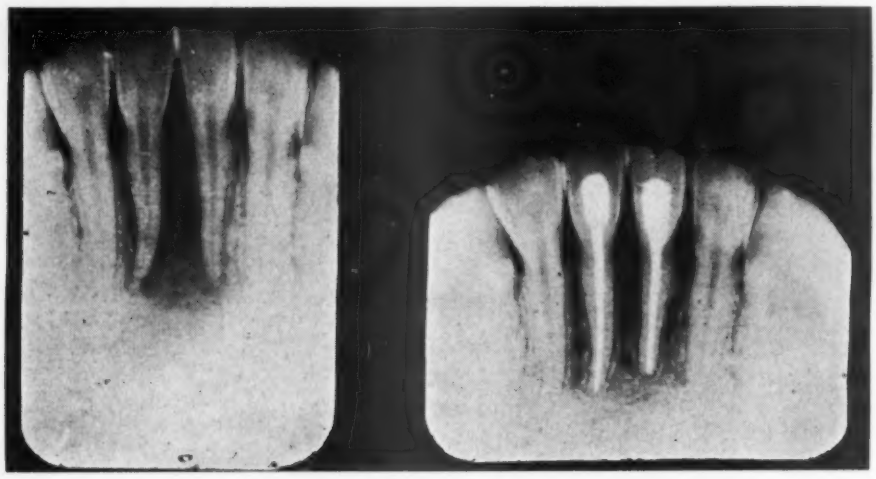


Fig. 14.

G. C. CHRONIC PROLIFERATIVE PERIOSTEITIS
Reg. No. 8067
Age 16

4/27/38	No organisms no cells	#24	No growth	4/29
4/27/38		#25	No growth	4/29
4/30/38		#24	No growth	5/2
4/30/38		#25	No growth	5/2
5/2/38		#24	No growth	5/4
5/2/38		#25	No growth	5/4

Fig. 15.

Formula for Dehydrated Phenolsulfonic Acid

Phenol	90 parts
Sulfuric Acid	97 parts

(Heat at 100° C. for 20 hours.)
Used in all canals prior to filling for root resection.

Fig. 16.

Owing to its powerful affinity for moisture, this solution, within a matter of a few minutes, will remove any residual organic débris remaining in the dentinal tubules. The acid has a thick syrupy consistency and may be conveniently carried into the canal on a No. 6 Kerr file. While the acid is in place, the walls of the canal may be honed to a glasslike smoothness. After neutralizing the acid with sodium bicarbonate in alcohol, the canal is dried with a hot Kerr plugger until all hissing ceases. Some operators advocate root resection prior to filling the canal, on the grounds that it is impossible to dry a canal sufficiently before filling. Our experience in thousands of cases does not coincide with this claim.

It should be emphasized that the acid is not intended for the chemical enlargement of the canal, but merely for the removal of foreign substances plugging the dentinal tubules.

GUTTA PERCHA TECHNIQUE FOR FILLING CANALS BEFORE RESECTION (FOR UPPER ANTERIORS)

Before attempting to fill the canal, the bracket tray should be so arranged as to contain only those instruments necessary for the operation of filling. Mynol fine, hand-rolled points are sterilized in metaphen germicidal solution, dried, and laid out on a sterile towel in a manner which will permit the operator to hold each point at a certain angle in the cotton pliers. This will permit greater ease in carrying them into place. Fig. 17 shows proper arrangement of materials. Small porcelain slab contains Kerr's Antiseptic Root Canal Sealer and Liquid.

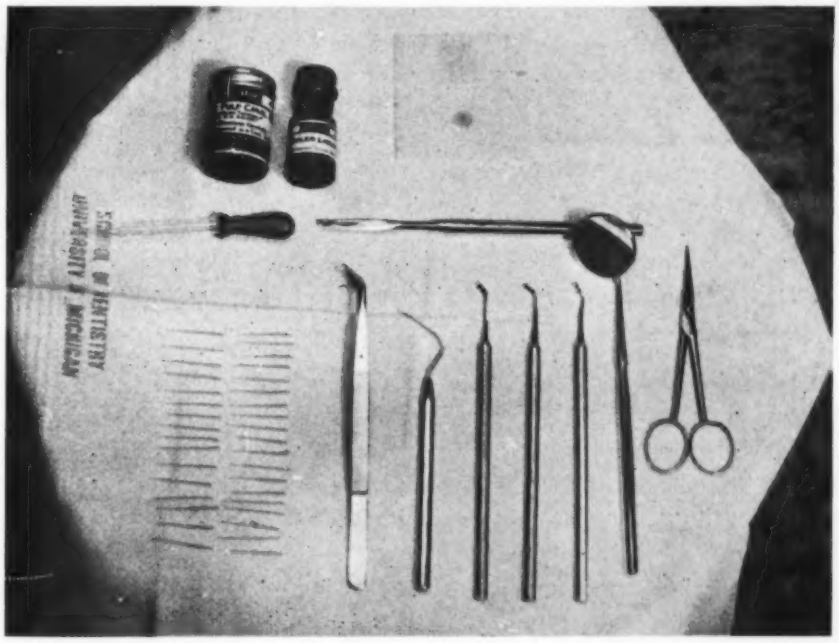


Fig. 17.

Sequential Steps.—

Select a trial gutta percha point which compares in length to that registered on History Chart and place into canal until large end is exactly even with incisal edge or tip of cusp. A radiograph taken at this time will indicate the accuracy of instrumentation. Corrections in length, if any, should be made at this time.

Mix Kerr's Antiseptic Root Canal Sealer on white porcelain slab from instrument case, using small Kerr spatula for mixing. Thorough spatulation of the powder and liquid are necessary in order to obtain a smooth homogeneous creamy mix.

*Formula of Kerr's Antiseptic Root Canal Sealer**

The composition of the set mass is:

Silver	24.74 per cent
Zinc oxide	34.00 per cent
Dithymoldiiodide	10.55 per cent
Oleoresins	30.72 per cent

*Dr. U. G. Rickert.

The working time of the above sealer is approximately ten minutes. This affords ample time for the filling operation.

Roll the trial gutta percha point in the Kerr sealer and carry it into place with a slight pumping motion so as to exclude air droplets that might have become trapped in the sealer.

Force the first gutta percha point to the side of the canal with a Kerr No. 3 root spreader and add a point each time the spreader is withdrawn. By using the spreader to sight through, and bringing the gutta percha point up parallel to the spreader, it is possible to insert the point each time into the hole made by the spreader the instant it is withdrawn. A slight delay means a loss of direction and recoil of the compressed gutta percha, making it practically impossible to get the point into place. Personal practice is necessary to master this particular step.

The average maxillary central incisor, lateral incisor, or cuspid will require from 15 to 25 gutta percha points in order to completely seal the canal. The diameter and taper of the points must of necessity be slightly smaller than that of the Kerr No. 3 spreader, or else they will not go into the opening made by the spreader.

The excess ends of gutta percha that protrude from the coronal opening of the canal after no further points can be added may be easily removed with a large straight *hot* spoon excavator.

By rotating a hot amalgam plugger into the canal, the gutta percha may be finished off to any level, leaving a clean flat surface.

MODIFIED TECHNIQUE FOR PERFORMING ROOT CANAL THERAPY AND RESECTION IN ONE SITTING

Because of the many emergencies in the Armed Forces that arose during the war, both the Navy and the Army, resorted to a shorter method whereby the entire operation could be performed in one sitting. We do not feel that this is the method of choice, and it should be used only where extraction would be the only alternative. The technique is identical to that previously described except that the preliminary treatments and cultures are eliminated. After thorough cleansing of the canal and proper instrumentation, the dehydrated phenolsulfonic acid is relied upon to bring about immediate sterilization of the canal and dentine. Consequently, at the time of resection, the operator is necessarily working in an area that may contain pathogenic microorganisms in varying amounts. Although too much emphasis, perhaps, need not be placed upon the potential dangers of postoperative infection from organisms in the periapical region, nevertheless it is advisable to attenuate whatever organisms may be present by the use of suitable germicides. Preparations containing sulfonamides have been advocated for use in the cavity following curettement as a precautionary measure against postoperative infection.

Because of the unfavorable healing following the use of sulfonamides in bone cavities, we have not been too enthusiastic as to their beneficial effects.

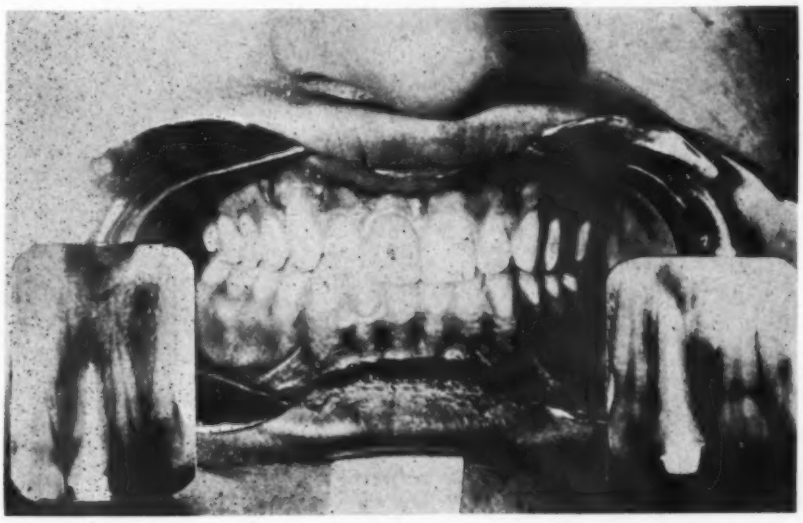
SILVER POINT TECHNIQUE IN TEETH TO BE RESECTED

Owing to the size and shape of the lower six anterior teeth, as well as the upper first and second premolars, it has been found more feasible to use Young silver points, which correspond in size and shape to Kerr files of the same num-

ber. The difficulty of enlarging the lower central and lateral incisor canals to a size necessary for adequate gutta percha compression will in most instances warrant the use of a simpler method. By the time these canals have been enlarged to a No. 4 file, it becomes apparent that the instrument fits the apical third very tightly. Whatever number file fits tightly determines automatically the size of silver point to be used. The same sealing material used in conjunction with gutta percha is also used with silver points. In the case of upper first and second premolars, a combination of silver points and gutta percha may be employed. The canals in the end third are almost identical in size to the lower anteriors, whereas the space between the buccal roots above the bifurcation may easily be filled with gutta percha.

NEED FOR COMPLETE HERMETICAL SEALING OF CANAL PRIOR TO ROOT RESECTION

In histologic studies of the root ends of resected teeth, Herbert[6] found that such inflammatory reaction as remains in connection with these teeth is strictly limited to the tissues immediately adjacent to the apical end of the root canal filling and is not found in association with the cut surface of the dentine, which shows evidence of repair over most of its surface. This suggests that so long as the porous apical third is removed, the risk of future infection after a root resection lies much more in leakage from the root canal than in retained infected dentine. If this is so, the success of the operation depends largely on obtaining an adequate seal with an impervious material for the apical end of the root canal.



After Fig. 18. Before

In Fig. 18 a large periapical involvement is noted on a tooth carrying a porcelain jacket crown. The tooth appeared to be well filled and was resected by the dentist who had inserted the crown. Two years later, the area appeared worse than before. Careful examination shows the canal has been filled with two gutta percha points affording ample leakage, and resulting in a loss of tissue repair.

TECHNIQUE FOR ROOT RESECTION

1. If history reveals a highly nervous patient, premedication with elixir of nembutal, taken one-half hour before operation, is deemed advisable.

2. Conduction anesthesia with 2 per cent novocain solution with suprarenin (1:50,000) properly given will usually suffice. If anesthesia is not complete, this may be supplemented with infiltration. Before injection, the tissues around the point of injection should be dried and thoroughly sterilized with tincture of mercresin.

3. A flap should be prepared large enough to give free access to the entire operative area. The line of incision should be at least ⅛ inch above the free margin of gum. When it becomes necessary to cross the median line, it is expedient to incise around the frenum, making somewhat of a V-shaped flap. This will serve as a convenient landmark for returning the flap to its original position.

4. In retracting the flap, a periosteal elevator should be used, carefully lifting the mucoperiosteum away from the cortical plate of bone without macerating the tissues. The more care exercised in manipulating the tissues, the less will be the postoperative pain and swelling.

5. The retracted flap may be held in place with the periosteal elevator. Do not exert unnecessary pressure upon the lip. This will minimize swelling.

6. If the periapical lesion has not perforated the labial plate, it is wise to explore for an opening with a cow-horn explorer, rather than to remove bone promiscuously with a chisel or burr.

7. When the periapical region has been located, the overlying bone, if present, can be removed either with a hand chisel or by careful use of the surgical burr.

8. A small curette is used to remove whatever pathologic tissue is present. This is placed in a specimen bottle containing formalin, for pathologic examination. Good hemostasis during the operation is important. Various types of suction devices for removing blood are in use and have gained more or less favor. The promiscuous use of gauze sponges pressed against the bone and into the cavity is irritating and will cause unnecessary trauma. By rolling the gauze into a small wick, it may be brought into direct contact with bleeding points, without irritation to the entire field of operation. Occasional flushing of the wound with warm normal saline solution will greatly minimize the bleeding and give a clear field of vision.

9. After most of the pathologic tissue has been removed, the root apex is merely smoothed off with a No. 560 straight crosscut burr. Contrary to general belief, it is not necessary to remove the root to the lowest point of pathology or to a level of sound periodontal membrane attachment. This constitutes an unnecessary sacrifice of sound tooth structure. The reattachment of the periodontal fibers will take place along any exposed surface on the side of the root as well as over the cut root end.

In Fig. 19 the periapical involvement extends well past the middle third of the root. Removal of the root tip was sufficient. Final radiograph seven years later shows satisfactory healing and normal attachment along the entire root surface.



Fig. 19.



Fig. 20.


May 13 1932
May 23 32
May 25 32
May 25 32
Dec 10-32
April 29 33
April 29 33
May 29 33

Fig. 21.

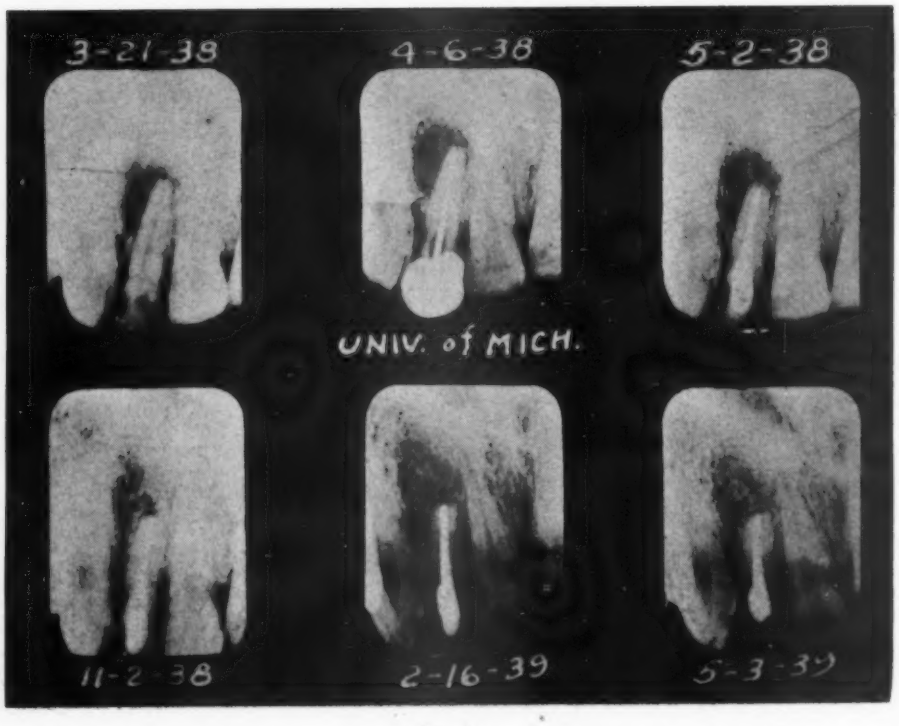

3-21-38
4-6-38
5-2-38
UNIV. of MICH.
11-2-38
2-16-39
5-3-39

Fig. 22.

In some instances the pathology extends down lower on one side than another. These may be resected on an angle to conform to the extent of pathology, as shown in Fig. 20, with very satisfactory healing.

10. The exposed surfaces of the root and smoothed apex are coated with a thin film of freshly prepared solution of ammoniacal silver nitrate, which, after remaining a few seconds, is reduced with eugenol or oil of cloves. In order to avoid spilling free silver nitrate onto the exposed alveolar bone, the silver nitrate is carried into place on a plain flat toothpick. The root end becomes coated with a black precipitate of silver.

The ammoniacal silver solution is prepared by dissolving sufficient $AgNo_3$ crystals in five or six drops of concentrated (28 per cent) ammonia in a dappan dish until a clear amber-colored solution is obtained and no precipitate follows with the addition of more $AgNo_3$.

11. The flap is carefully returned to place and the wound closed with interrupted sutures of black cotton or silk, using a curved needle. Sutures should be evenly spaced, about $\frac{1}{8}$ inch apart, in order to insure perfect adaptation of the flap.

12. Suturing completed, the area is painted with compound tincture of benzoin. This serves both as a protective coating and an antiseptic. If the foregoing steps have been carried out as directed and with careful manipulation of the tissues, there will be in most cases practically no postoperative swelling, and little if any postoperative pain.

13. A carefully timed radiograph is made, recording the exact angle and exposure on the film mount. Subsequent radiographs made under the same exposure factors will give an accurate record of the rate of bone calcification.

14. Sutures may be removed in three to five days, using a small shears with sharp points.

Figs. 21 and 22 show checkup radiographs properly mounted in a convenient type of mount, showing the dates when films were taken. In such a series the operator can see at a glance the progress of the case.

ROOT RESECTION IN PERIAPICALLY INVOLVED TEETH CARRYING CROWNS WITH POSTS OR RESTORATIONS, MAKING PREVIOUS ROOT CANAL THERAPY AND FILLING IMPOSSIBLE

Quite frequently we find teeth with good porcelian jacket crowns reinforced with gold cores, Davis crowns, or Richmond crowns, in which root resection is indicated. However, the removal of the restoration for the sake of filling the canal would entail such difficulties as to make the entire effort unwarranted. The periapical tissues in these cases may be restored to normal without disturbing the restoration in any way.

Sequential Steps.—

1. An accurate radiograph is essential.

2. The operation is identical to technique previously described, up until the time the root tip is localized.

3. Completely expose the apical third of root.

4. With a No. 560 crosscut burr held in a straight handpiece, remove apical root tip at an angle pointing toward the incisal edge so as to permit a better view of the canal opening.

5. The apical portion of the canal may now be enlarged by means of Kerr files having a right-angle bend ¼ inch from point of file.

6. Rotate the file in a semicircular arc, at the same time pressing the point down into the canal with a flat instrument.

7. Enlargement of the apical portion of the canal should be carried to a size where the file fits the canal with the accuracy of a closely fitting piston. It is important to note that the same principle is employed as that previously advocated for silver points in lower anteriors, namely, the canal assumes the same size and shape as the instrument which cuts it.

8. A ¼ inch section of a silver point corresponding in number to the last file used is now fitted into the canal in such a manner as to enable the operator to withdraw it. In other words, the silver point should extend about 3 mm. beyond the cut surface. (Fig. 23.)



Fig. 23.

9. Before sealing the point into place the canal is sterilized by carrying dehydrated phenolsulfonic acid into place on the last file used. After neutralizing with sodium bicarbonate, the canal is dried with a hot Kerr plugger bent in a manner similar to the files.

10. The selected section of silver point is now rolled in a rather thick mixture of Kerr Antiseptic Root Sealer, carried into place, and firmly locked by tapping with a flat chisel.

11. The excess silver point that had protruded from the canal is now smoothed off even with the root end. If burr is revolving clockwise, care must be used to start cutting from left to right, in order to prevent the revolving burr from pulling the point out of the canal.

12. The reduction of silver nitrate, subsequent suturing, and painting field of operation with compound tincture of benzoin complete the operation.

Advantages of Above Technique.—

1. Pathologic conditions involving teeth carrying Davis crowns, porcelain jacket crowns with gold cores, Richmond crowns, or ¾ restorations with posts may be corrected without necessitating the removal of the restoration.

2. There is no difference in the healing time in this method as compared to the usual procedure.

REQUIREMENTS FOR SUCCESSFUL BONE REGENERATION (FOLLOWING APICOECTOMY)

1. *Blood Vessels.*—There is an exceedingly rich blood supply in the cancellous portion of the mandible and maxilla which amply provides for the first requirement in osteogenesis.

2. *Building Material.*—Immediately following the operation of root resection the small cavity becomes filled with blood which coagulates rapidly and becomes invaded by fibroblasts and angioblasts, which leads to its organization and transformation into young connective tissue. In short, a young connective tissue fills this cavity, which is the building material, and all of the conditions for osteogenesis are present.

3. *Stimulus.*—The stimulus is provided for by the trauma that is produced by the operation. It is a well-known fact that any irritation to bone tissue is a stimulant to osteogenesis. After the operation the mucoperiosteal flap is returned to its normal position and sutured, and we have all of the required elements present for the repair of bone.

Postoperative Repair.—The histologic changes occurring around the periapical region of pulpless teeth have been adequately described by Edgar Coolidge. In his book entitled *The Clinical Pathology and Treatment of the Pulp and the Periodontal Membrane,* he describes the reparative stages as follows: "An initial period of inflammation is followed by a period of rapid resorption of the necrotic soft tissue during which even some of the hard tissue of the tooth may be absorbed. A state of equilibrium or tolerance is finally reached and repair begins, circulation decreases, fibroblasts enter, and cementoblasts repair the resorption in the dentine, reducing the size of the apical foramen with secondary cementum which also covers resorption in the old cementum and in the surface of the root."

Many cases have been reported where the apical foramina have been completely sealed by this fresh tissue, termed by E. Wilfred Fish[7] as calcific tissue of repair. Thus it is possible for the remaining dead dentine in a pulpless tooth to be isolated completely from the surrounding tissue by a barrier of healthy, living cementum. It is for the same reason that good tissue repair will occur following simple smoothing of the root during root resection, rather than amputation down to the lowest level of pathology.

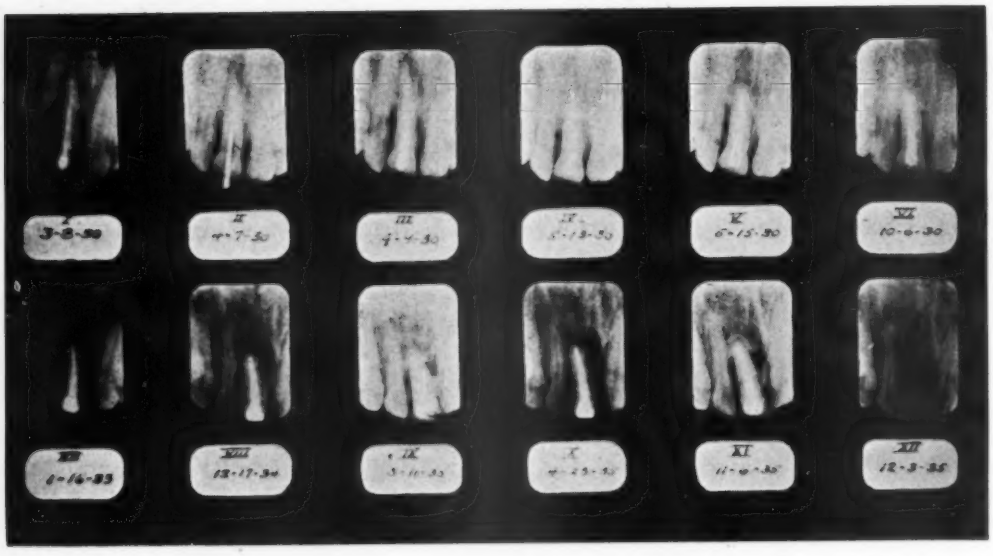


Fig. 24.

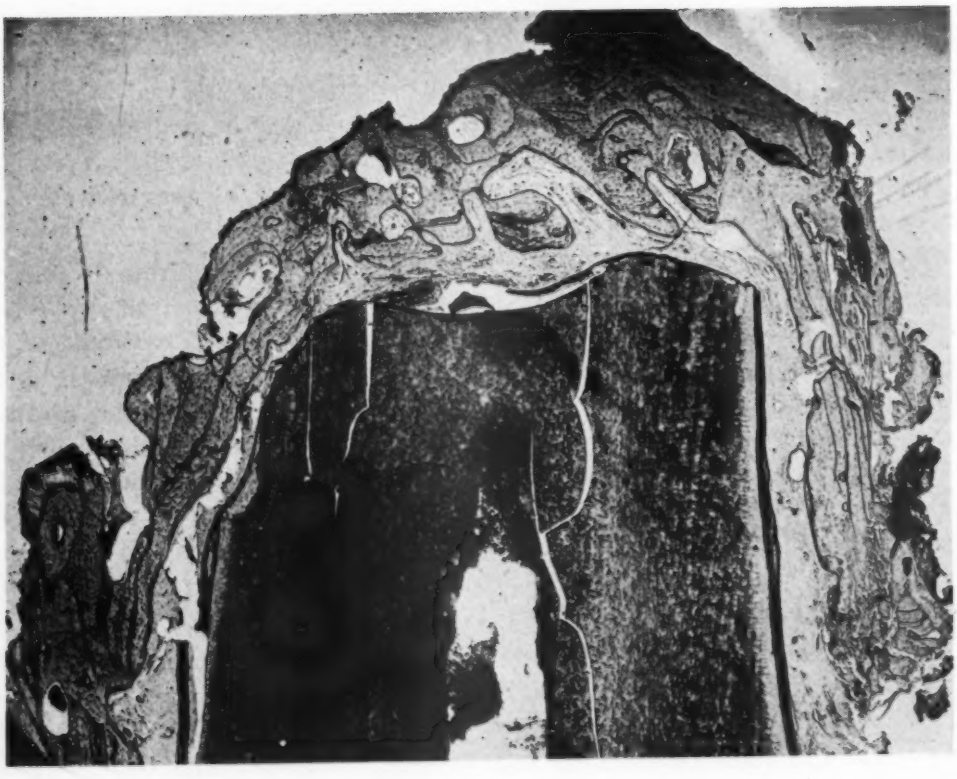


Fig. 25.

Cases have been reported by various authors indicating that a similar tissue reaction will take place under favorable circumstances after root resection. Coolidge reports one such case where histologic examination was made fourteen years after root resection. This showed complete repair with new cementum attached to the entire cut surface of the dentine, the cut end of the gutta-percha root filling being in opposition with a band of fibrous tissue attached on either side to this new cementum.

Fig. 24 is that of a 17-year-old boy with a fractured central incisor. At the time this patient entered the clinic, the radiograph revealed some periapical involvement, as well as what appeared to be a slight pericemental abscess.

Routine root canal therapy and resection was performed, followed by periodic radiographs (Fig. 25).

From the series it will be noted that the periapical tissues returned to normal. What appeared at the time to be a pericemental abscess has since envolved the entire labial plate of bone. It is quite apparent that at the time of fracture of the crown, the labial plate was injured, resulting in gradual bone necrosis. Since extraction of the tooth was warranted on the grounds that the supporting bone was being destroyed, the patient consented to have the tooth removed surgically, enabling us to secure the alveolar bone surrounding the apical area. The histologic section of this tooth shows deposition of cementum over the cut root end, surrounded by a normal thickness of periodontal membrane and normal bone architecture.

In order to further substantiate the health of the regenerated tissues following root resection, W. E. Herbert[8] of Canada reports bacteriologic examinations of resected teeth after complete healing had taken place. His method was to pack off the area with sterile gauze sponges and apply iodine to the surface of the mucous membrane for one minute. A sterile swab moistened with broth was then taken from the surface of the old operation scar. A sample of the local anesthetic solution was then injected into tubes of broth and a submucous injection of a few minims immediately made. The root resection scar was then touched with the actual cautery and a sterile burr inserted into the region of the apex, care being taken not to generate heat. Material from the burr was then collected and cultured in broth both aerobically and anaerobically. Of twelve cases tested, only two showed any growth, and these were clearly air-borne contaminants.

Extensive Bone Involvement.—All too frequently, periapical pathology has progressed to a point where several teeth appear to be involved. Clinical experience has shown that the radiograph generally minimizes the actual extent of pathology. If the area of radiolucency comes into close proximity with sound teeth, there is a good possibility of destroying the blood supply of the teeth during the process of curettement. In such cases, it is wise to include the adjacent teeth in the operation, as shown in Fig. 26. Although the laterals were vital at the time, it was felt safer to include them in the resection rather than risk the possibility of a failure through subsequent pulp death following the operation.

Age does not appear to influence the subsequent repair. Fig. 27 shows complete repair, after eleven months, of an upper cuspid in a man aged 59 years.

Extensive lesions of the maxilla will show good repair provided the palatal plate is not destroyed. If and when it is, an operative defect usually appears at the approximate center of the original site of the lesion. Fig. 28 shows a complete bone matrix in fourteen months following resection of a central and lateral incisor with an extensive pyogenic cystic involvement.

STUDY OF THE PATHOLOGIC CHANGES FOUND IN 47 CASES OF ROOT RESECTION*

The pathologic changes observed in the tissue removed at the time of root resection are of a uniform character and are indicative of a chronic inflammation. The variations in inflammatory response can be placed in three distinct categories.

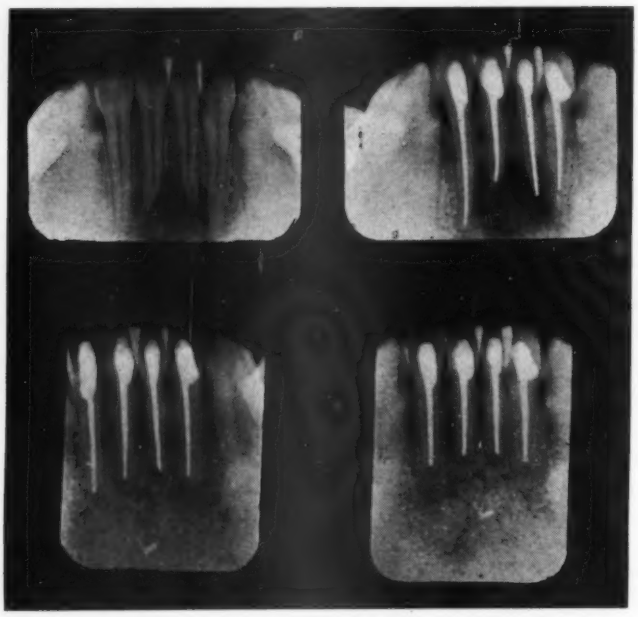


Fig. 26.

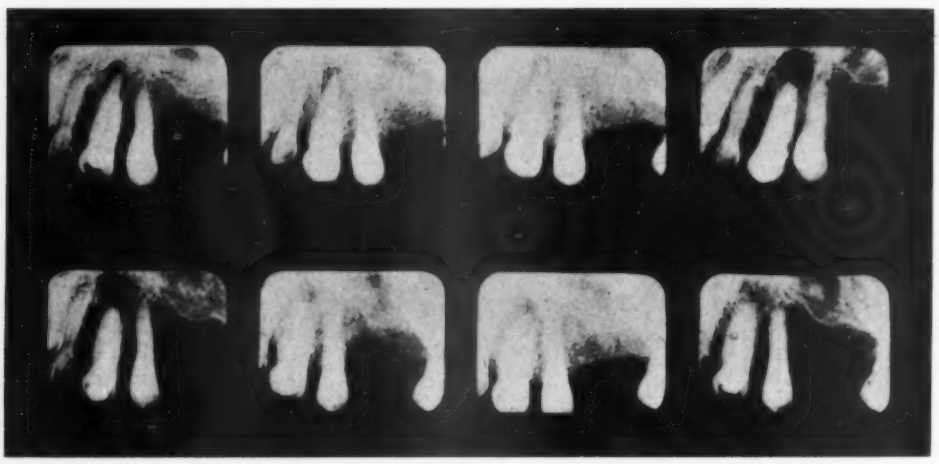


Fig. 27.

Group 1.—In 29, or 62 per cent, of the resected cases, the inflammatory response was characterized by the presence of a chronic vascular granulation tissue.

Group 2.—In 6, or 12.8 per cent, of the tissue removed at the time of resection, there was evidence of a chronic abscess, the wall of which was composed of vascular pyogenic granulation tissue.

*Made and reported by Dr. D. A. Kerr of the Department of Pathology, School of Dentistry, University of Michigan.

Group 3.—In 12, or 25.5 per cent, of the cases, there was vascular pyogenic granulation tissue present. Alveolar process included in the material exhibited changes typical of chronic productive osteomyelitis.

In Group 1, the inflammatory reaction is not specific for any particular etiology. The tissue changes are fibroblastic and angioblastic proliferation resulting in the production of an immature vascular connective tissue, in which inflammatory cells of all types are found, the predominant cell being the large mononuclear cell. In lesser numbers are the lymphocytes, plasma cells, and the polymorphonuclears. In those instances where epithelial rests are present in the periodontal membrane, the epithelial cells also show evidence of proliferation. This type of reaction is not characteristic of any particular etiology and could be produced by pulp degeneration with or without associated infection. These changes are indicative of only a chronic inflammatory process localized to the periapical tissues.



Fig. 28.

In Group 2, the same type of granulation tissue is present and forms the wall of a chronic abscess cavity. The presence of pus is indicative of a pyogenic process which is usually due to a pyogenic type of microorganism. The presence of the granulation tissue surrounding the abscess is evidence of its chronic nature, and indicates the ability of the patient to localize infection. Findings of this character suggest a pyogenic etiology and a high degree of resistance to infection which results in localization to the periapical tissues.

In Group 3, the changes are more extensive. There is a chronic purulent inflammation of the periapical tissue with direct extension into the surrounding alveolar process. The narrow spaces are filled with a vascular granulation tissue which is rich in polymorphonuclear cells. This is indicative of a chronic productive osteomyelitis. These changes show that the process is of a pyogenic nature and of such a severity that it was not confined by the body to the periapical area. (The incidence of localized osteomyelitis might have been higher in this series, if bone had been examined from every case.)

SUMMARY

1. Whenever pulp disease has progressed to a point beyond the dentocemental junction, a twofold problem is presented, namely, a root canal problem and a bone problem.

2. The root canal problem must be managed in a manner so as to leave no doubt that every vestige of irritation arising from within the canal has been definitely and permanently eliminated.

3. The bone problem consists in the removal of any and all pathology beyond the root end and must be accomplished in a surgically clean and thorough manner.

4. If and when both phases of the process have been so executed, the return of the tissues to a normal physiologic state will inevitably follow, provided the physical and clinical conditions have warranted the procedure.

5. The pathologic findings in Groups 2 and 3 support the rationalization that sterilization of the tooth previous to resection is the treatment of choice.

6. If 38 per cent of the 47 cases in which pathologic studies were made show evidence of either a chronic abscess or a localized osteomyelitis after treatment, certainly a higher percentage would show the same changes before treatment, and the changes would no doubt be of a more severe and active nature. Treatment and sterilization of the tooth previous to resection reduces the infection and permits the body defenses to localize the inflammatory process and reduce its activity. Because of attenuation of the organisms, decreased severity, and localization of the infection following treatment, there is less opportunity for infection to be spread by surgical manipulation. Healing and repair will, therefore, take place more rapidly. With a decrease in severity and a localization of the infection, postoperative complications will be reduced to a minimum.

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PRINCIPLES AND TECHNIQUE OF THE REMOVAL OF MOLAR TEETH

PHILIP M. NORTHROP, D.D.S., M.S.*

MANY articles have been written discussing the management and technical procedures for the removal of the maxillary and mandibular third molars. Such an abundance of literature on this subject is to be expected, since the problems arising from the third molar and the technical difficulties encountered in its removal are some of the most difficult of all oral surgical operations.

In the present day of specialization, difficult third molar extractions are usually referred to the exodontist or oral surgeon. Such a disposition of the patient is logical for several reasons.

First, the exodontist or oral surgeon is better prepared through experience and training to diagnose, treat, and assume the responsibilities accompanying their management. Since he constantly is confronted with this problem, it is reasonable to assume that he should be able to render a better service to the patient than the dentist who has had very little surgical training, or one who may have occasion to remove this tooth only at infrequent intervals.

It is difficult, in addition, to estimate accurately how much time such a procedure will entail. If the general practitioner has a full operative schedule of appointments for the day, and difficulties occur in the removal of such teeth, as often is the case, overlapping of appointment hours is apt to occur. As a result, a smooth-running operative program may be greatly interrupted. Further, when one becomes aware of a crowding of time, there is a tendency on the part of the operator to hurry. Hurried operating will involve unnecessary tissue injury, with increased mental and physical tension. Such an experience tends to fatigue the operator, and at the same time the operation terminates in an unsatisfactory way.

It has been my own observation that the general practitioner often will accept all of the disadvantages and responsibilities of a surgical problem and not secure a satisfactory or reasonable financial remuneration for his service. He cannot be adequately compensated unless a fee is charged which usually seems to the patient out of proportion to the service that actually was rendered.

It is not my desire to caution the general practitioner in order to stimulate the referral of patients with surgical problems to the oral surgeon or exodontist. Many general dental practitioners perform excellent surgical service and should continue to do so. It is rather an attempt to caution the dentist to hesitate before acting; to urge careful and thorough diagnosis and to outline a treatment pro-

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cedure which will take into consideration complications that may arise. The young dentist is urged to avail himself of further surgical training in the many postgraduate courses now being presented in dental schools. One should select the more simple surgical cases at first, and then, as more experience and confidence are gained, attempt the more difficult types of operations.

It is my conviction that the average general practitioner would profit when he recognizes the difficult surgical problems if he would refer them to one more skilled in their management. He would relieve himself of added responsibility and nervous strain which such surgery entails and would not disrupt a smooth office routine. Not only is this statement true for the treatment of the third molar, but it applies also to any difficult surgical procedure which requires unusual diagnostic and technical experience or skill.

It is primarily the purpose of this paper to discuss the removal of difficult molars other than the third molar. Little has been written relative to this important phase of exodontia. Frequently, dental literature stresses the unusual and spectacular procedure when there is an urgent need for information and knowledge about everyday "bread and butter problems." Often these problems are difficult to handle and the practitioner is apt to be confused, since their management frequently is passed off by others as a simple procedure.

The removal of difficult first and second molars presents problems which need discussion. I am sure that any experienced exodontist often would much prefer to remove a difficult impaction than many first and second molars. In the case of an impacted tooth he immediately realizes that he must, to some extent, be radical in his methods; whereas in the removal of the nonimpacted tooth, the tendency is to be conservative. As the procedure progresses he becomes more and more radical and at the termination of the operation the tooth has been removed piece by piece. As a result the surgeon as well as the patient is tired and disgusted. Further, he thinks that he has been exceedingly crude and inefficient in his surgical judgment and skill. Such a response is only normal and yet not necessarily justified, because the extraction of any tooth can be a most trying procedure and may require extreme measures.

Realizing these surgical difficulties, this article is written, then, to present some of the principles and techniques, which, if utilized, will be most helpful in the removal of these molar teeth.

DIAGNOSIS

The problem of diagnosis itself is a subject which demands much discussion. Space will not permit such a discussion in a short paper except to stress the importance of diagnosis and urge a careful study of the patient. Following the diagnosis, one should plan thoughtfully the best method of operation. This latter is very essential and will aid tremendously in obtaining a satisfactory end result. Careful treatment planning will give the operator a full appreciation of the extent of the surgery required and the complications which may be encountered.

RADIOGRAPHIC STUDY

There can be no question that the proper use of radiograms is one of the greatest aids to the dentist in arriving at a correct diagnosis. Although preoperative radiograms are advisable for all extractions, they are particularly necessary when molar teeth are to be removed. Many dentists, although admitting its soundness theoretically, have the opinion that such a routine procedure is unecessary and that they do not have time in a busy practice to do it. Actually he is not losing time in waiting for the development of a dental film if his time is organized properly. If the film is exposed before the administration of the local anesthetic and developed immediately, it will be available by the time the anesthetic has taken effect.

Securing good dental films prior to surgery, particularly of any molar extraction, is most essential, since the method or technique of removal depends to a great extent on the radiographic findings. Without films which show the tooth form and the density of the surrounding bone, etc., one certainly is inviting trouble. The patient has been so educated today that he expects dental x-rays to be made. If they are not taken, there is often the question of operator inefficiency in the patient's mind. It is no longer necessary to justify to the patient the need for dental films.

The question frequently arises about a fee being charged for x-rays taken at the time of extraction. It is my feeling that no separate fee should be asked. An x-ray is an aid in diagnosis and treatment planning and the charge for it should be included as part of the cost of service. Such a routine is not only essential to the surgeon but also has a favorable psychological effect on the patient.

ASEPSIS

One of the cardinal surgical principles that should be followed closely is an aseptic technique. All dentists recognize its importance, and yet it frequently is not observed, due either to carelessness or ignorance. Many of the severe deep infections of the face and neck are the result of the introduction of pathogenic organisms. Often a needle is used for a deep injection immediately following its use in a contaminated area. It is always essential to use a sterile needle, particularly for a deep injection such as the zygomatic and inferior dental block, and not one previously used for another injection on the patient.

Before injection, the field should be wiped dry with sterile gauze and painted with an accepted effective germicide. After this procedure is carried out and before injection is made, one should not permit the patient to contaminate the sterilized site by closing the mouth or allowing it to come in contact with some other contaminated surface. The drying of the site of injection before the application of the germicide insures more complete sterilization of the mucous membrane. If the germicide is painted on a field covered with saliva, it is doubtful if it actually contacts the mucous membrane and hence can exert little or no sterilizing action. Another break in technique, permitting the introduction of pathogenic organisms, occurs when the inferior dental block is

made and the needle is allowed to contact the lateral surface of the tongue. This means of contamination is greatly increased in the patient who has a small mouth or a large active tongue. Although the site of injection may have been sterilized, yet if the lateral surface of the tongue or any other oral structures are contacted with the needle, it becomes contaminated and pathogenic organisms may be carried into the tissues. It often is helpful in this injection to curve the needle by bending it on sterile gauze. A slight curvature of the needle permits the injection to be made from above the tongue and from the anterior position, without contacting other unclean surfaces.

ANESTHESIA

Another important prerequisite for a successful surgical procedure is satisfactory anesthesia. In this instance reference is made to the use of local anesthesia. If a profound anesthesia can be obtained and the patient is shown that the sensation of pain has been eliminated, immediately he is relieved of his anxiety and fear. The surgeon also is not under tension and can proceed with the operation with confidence and deliberation. There is no greater aid to the surgeon in the accomplishment of a successful surgical operation than complete anesthesia.

For many years it has been my desire to produce anesthesia with the minimal amount of anesthetic solution. This unquestionably is a conservative and safe practice. It is true that if an anesthetic solution can be deposited at the desired location a large quantity of anesthetic solution is not needed. This is demonstrated clinically when a nerve is contacted during injection and the patient experiences a sharp pain in the structures innervated by the nerve. Usually anesthesia occurs very quickly and is of a profound character because the solution is deposited directly around the nerve trunk.

Certainly the minimal amount of anesthetic solution should be used when dealing with a hypertensive and the "cardiac" patient, a hyperthyroid and extremely nervous individual, or with the patient presumably sensitive to a local anesthetic solution. A small amount of anesthetic solution is indicated in order to minimize the stimulating effect of the vasoconstrictor contained in most anesthetic solutions.

In making any injection, and especially in the type of patients just mentioned, it is desirable to deposit the anesthetic solution slowly, exercising great care that the anesthetic is not deposited directly into a blood vessel. Injection into the blood stream occurs more frequently than is suspected, especially in the zygomatic and inferior dental block. If the patient feels a sharp stinging sensation after the initial entrance of the needle, one should suspect the penetration of a vessel. In all deep injections and those in which this sign occurs, the plunger should be drawn back in the syringe. If blood flows into the syringe it is advisable to withdraw the syringe and reinject with another syringe. It is well to remember that there is a marked increase in toxicity of the anesthetic solution if it is deposited into the blood stream. If injected into a vein the

toxicity is increased from ten to twenty-five times, while in the arterial system the toxicity increases about four times as compared to a subcutaneus injection.

A patient lying in a prone position has less cardiac stress and can withstand a local anesthetic much better than in a sitting position. A patient subject to syncope, usually psychological in nature, often can be injected without the usual reaction if lying down.

Exclusive of the above type of patients, larger qantities of the local anesthetic can be safely used. It has been my experience, when so doing, that more profound and lengthy anesthesia is assured.

ACCESSIBILITY

One of the most important principles of surgical technique, but one which frequently is not observed, is adequate accessibility to the operative field. In observing the surgical technique of many postgraduate students during the past ten years, I have noticed that this fundamental principle was the one most often neglected. The common tendency is to be too conservative. The operator rather than reflect the adjacent soft tissue attempts to operate without a clear view of the field. As a result the soft tissues are repeatedly traumatized. The underlying bone and tooth structures cannot be viewed properly, trauma occurs, and the operative time is increased. If however, the tissue is reflected so as to give a good visual field, injury is minimized and the surgical procedure can be terminated more quickly. This technique, of course, reduces postoperative swelling and pain and promotes more rapid healing. A word of warning should be introduced at this point. There is the tendency on the part of the inexperienced operator, when he first makes tissue flaps or incisions, to go to the other extreme.

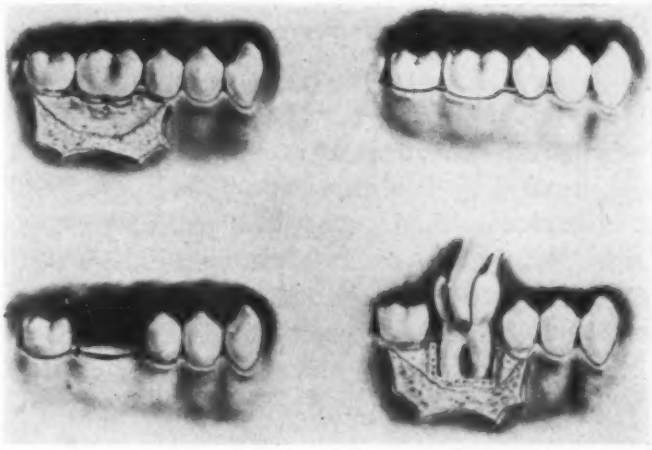


Fig. 1.—Showing accessibility to operative field by reflection of a gingival mucoperiosteal flap.

One can be too radical as well as too conservative. Accessibility should be planned by creating soft tissue flaps which will give optimum visibility for the requirements of the surgical procedure, but not so extensive that unnecessary trauma is done. As illustrated (Fig. 1), accessibility can be accomplished merely by reflecting a gingival mucoperiosteal flap. The flap is prepared by

first incising with a sharp knife the interproximal soft tissue both to the distal and mesial of the tooth to be operated. A blunt periosteal elevator is then carefully carried under the gingival papilla beneath the periosteum, otherwise the thin mucous membrane may be torn. If the instrument is carefully inserted beneath the periosteum, then the flap can be reflected cleanly and quickly from around the neck of the tooth and crest of the alveolar ridge to any height or depth desired. Care should be taken to strip away the periosteum only as far from the gingival margin as is required in order to give proper exposure. One should beware of retracting the periosteum from the bone too extensively, especially in the anterior part of the mouth and along the sides of the mandible. In the former, loss of the height or depth of the labial fold will result, while in the latter, marked tissue reaction may ensue due to the extravasation of tissue fluids beneath the periosteum.

Aspiration of blood and saliva is an important aid to the operator in all surgical procedures by giving a good vision of the operative field. It also stimulates bleeding of some of the bone surfaces and may be an advantage in preventing a "dry socket," especially in lengthy operative procedures. It is well known that often, after operating for several minutes, bleeding stops, and it is difficult to obtain a good flow of blood that will fill and clot in the socket following the operation.

It is desirable, following the extraction, to remove carefully all particles of débris from the operative field. Suction is extremely beneficial for this purpose. Where large flaps are prepared, if suction is not used it is necessary frequently to irrigate the field. With a large exposure and the constant expectorating by the patient, occasionally a negative pressure is created beneath the flap, resulting in air being sucked beneath it, causing an emphysema of the tissues of the face.

INCISIONS AND FLAPS

The use of incisions to give adequate exposure often is neglected when needful and, when employed, may be greatly misapplied. Neglect in its use when required results in trauma. Its abuse results from incorrect planning or a too radical procedure. Many times an incision is made directly over the operative field. In the course of the operation the margins of the soft tissue as well as the bone beneath the incision are traumatized or removed so that when the flap is returned to its normal position and sutured it does not have a firm bony framework on which to rest. Frequently the margins of soft tissue separate and collapse into the socket, exposing the bone. This results in prolonged and painful postoperative convalescence and many times leaves an undesirable deformity. A sound surgical principle to keep in mind, if an incision is made, is to design the incision at least one tooth beyond the operative field (Fig. 2).

The incision should be made by carrying the blade of the knife through the mucous membrane, submucous tissue, and periosteum in one firm and deliberate sweep, while holding the tissues taut over the underlying bone.

Incisions frequently are used when adequate exposure can be obtained merely by detaching and reflecting the gingival tissues adjacent to the operative field. This procedure is the one that is recommended; in fact, it is seldom necessary to use a vertical buccal or labial incision. Such a technique will give surprisingly good visibility and the flap automatically returns to its normal position and will heal readily without noticeable scar or deformity.

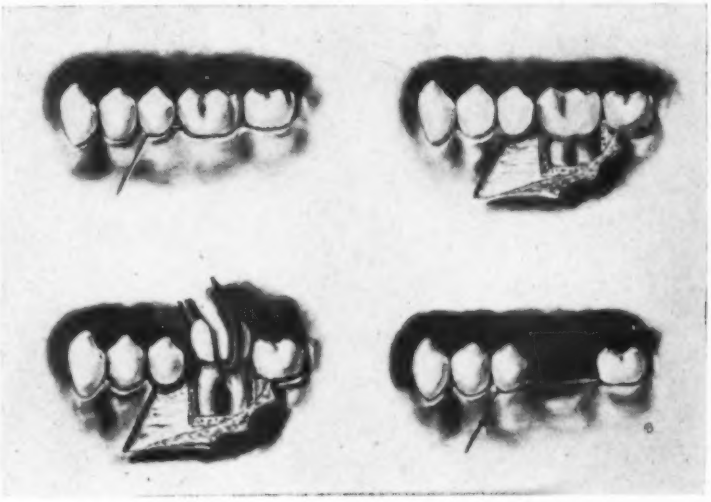


Fig. 2.—If oblique incision is prepared, it should be designed so that it is made at least one tooth beyond the operative field.

EXTRACTION OF MAXILLARY MOLARS

Although the discussion presents a plea for the use of mucoperiosteal flaps in order to obtain good accessibility, it should not be concluded that this technique should be employed as a routine method in all cases. One always should remember that the most desirable and successful procedure is the one which accomplishes the best end results with the least possible injury to the tissues.

In a large percentage of cases, after careful clinical and radiological studies, it is evident that extraction can be accomplished readily by simple forceps technique. Even after simple extractions with forceps the margins of the bone are often sharp and prominent or the soft tissue margins are adherent or irregular. For this reason the gingival tissue should be elevated from the crest of the ridge to a point at or beyond the prominence of bone around the neck of the tooth. Following the extraction the bone margin should be smoothed by the use of a rongeur or bone file. If the soft tissue readapts itself to its original position no suture is necessary, otherwise suturing is indicated.

If the tooth cannot be removed with the application of reasonable force, one should retract the buccal soft tissue to a point where adequate accessibility and exposure is obtained. By the use of rongeurs, chisels, or bone drills (the selection being an individual choice depending on experience and training), the buccal plate is removed just under the bulge of bone found around the neck of the tooth. Many times this slight relief of bone is sufficient to permit ready extraction. (Fig. 3.)

If removal still cannot be effected, or if the difficulty is well known by previous studies, two methods are advocated which will permit the removal with safety and minimal trauma.

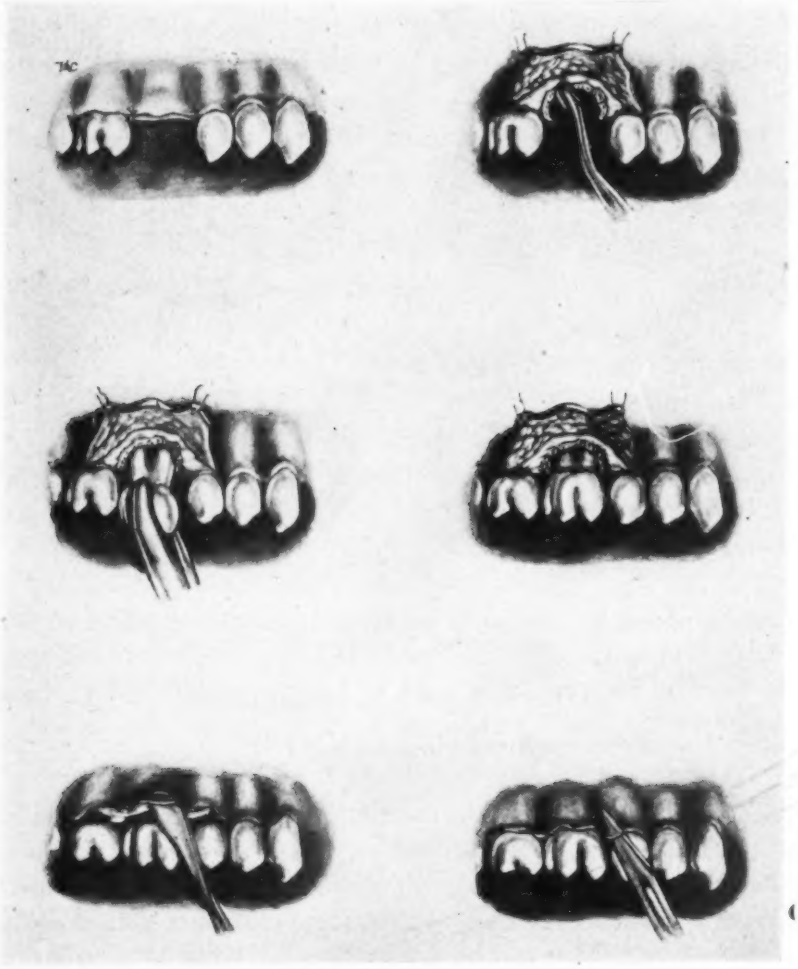


Fig. 3.

SECTIONING THROUGH THE CEMENTOENAMEL JUNCTION

The first method utilizes the fact that at the cementoenamel junction the pulp chamber is of its greatest diameter and here fracture of the crown from the remainder of the tooth can be accomplished easily and quickly (Fig. 4).

After the soft tissue has been reflected and the bone removed slightly above the bifurcation of the roots, it is desirable to obtain some movement in the tooth. This movement usually can be accomplished to some slight degree. This movement in the tooth is important, for it assures a separation of the roots from their periodontal attachments. Later, after the roots are divided, their removal will be greatly simplified. With the tissue well retracted a small surgical drill is carried through into the pulp chamber from the buccal aspect, over each buccal

root at the cementoenamel junction. These punctures are then joined, creating a small pathway into the pulp chamber. A small flat elevator is inserted into this groove. If this instrument fits snugly, so that there is no lateral movement, a firm twist of the wrist will produce a clean fracture through the entire crown. With the same surgical drill the three maxillary roots can be readily sectioned. Since movement previously has been established, removal of these roots usually can be accomplished readily by the use of elevators or small root forceps.

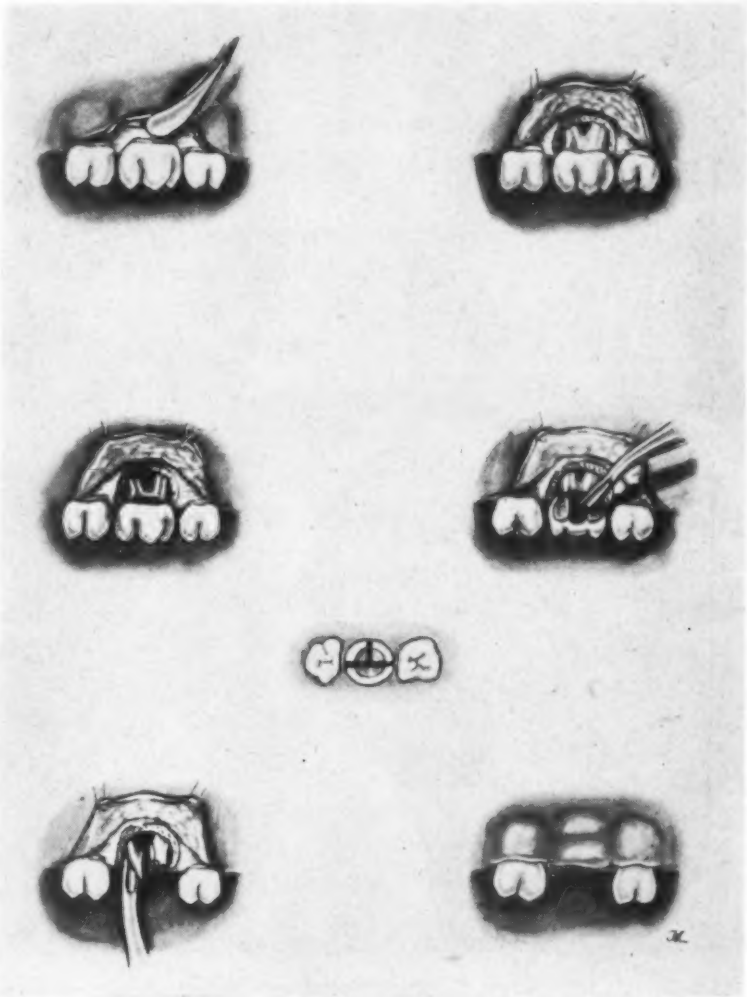


Fig. 4.—Sectioning crown of tooth through cementoenamel junction. Sectioning of three roots with their subsequent removal.

The second method will facilitate greatly the removal of these molars, permitting the lingual root to be removed with the crown of the tooth after the crown has been sectioned from the two buccal roots. This procedure is indicated particularly where the tooth has a well-constructed crown (Fig. 5).

Identical steps are carried out as in the previous method up through the point where exposure and movement in the tooth have been obtained. Instead

of sectioning at the cementoenamel junction, each individual buccal root is separated from the crown. The crown and lingual root then are extracted with a forceps and the loose buccal roots removed with an elevator or small forceps. Often there is only the need of sectioning one of the buccal roots from the rest of the tooth, especially if it is found to be markedly curved or widely divergent.

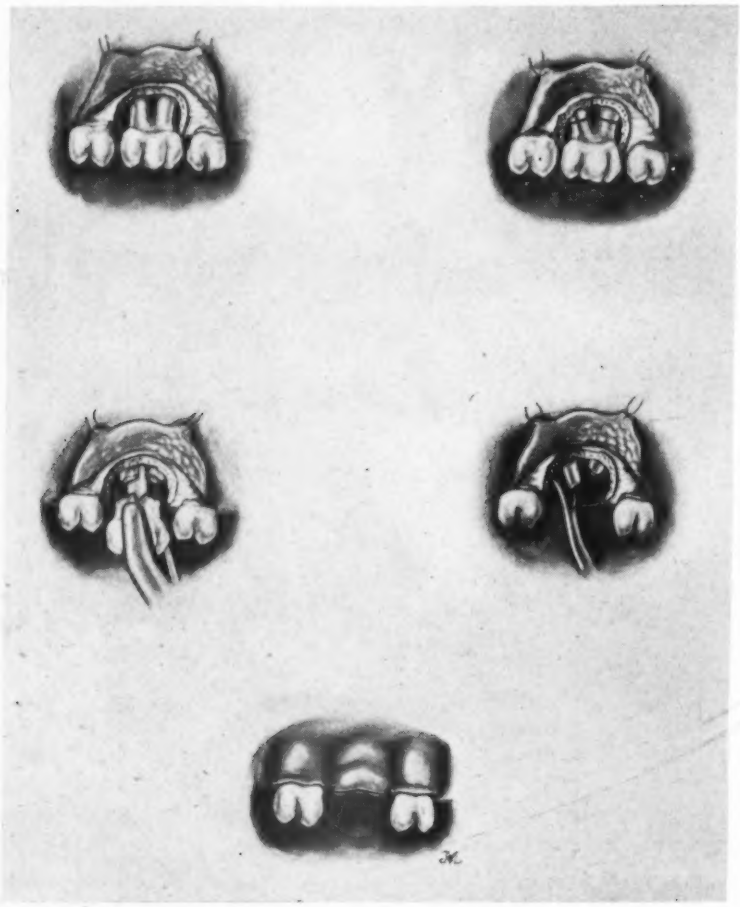


Fig. 5.—This method shows sectioning of two buccal roots with the removal of the crown with the lingual root intact.

FRACTURE OF MAXILLARY TUBEROSITY

These same procedures are recommended especially where there is the possibility, during the extraction of a maxillary molar, of fracturing the tuberosity of the maxilla. Fracture of a portion or the whole of the tuberosity may occur easily, often when very little force has been exerted during extraction (Fig. 6). This accident occurs because the teeth are large and have widely divergent roots, while the buccal bone around the neck of the tooth is heavy and the bone higher on the roots is very thin. This condition is encountered when a large maxillary sinus is present. Recognition of these abnormalities and the application and use of such techniques as have been presented here will prevent such undesirable complications.

EXPOSURE OF THE MAXILLARY SINUS

Frequently during extractions of the upper premolars and molars the maxillary sinus is exposed or penetrated. For some reason or other, when this exposure occurs, many operators deem it necessary to irrigate, pack, or enter such an opening. When the maxillary sinus is encountered it is well to keep away from it. Packing or irrigating such an opening increases the incidence of maxillary sinusitis. If a portion of the tooth has entered the sinus, its removal is indicated; otherwise, every attempt should be made not to enter the sinus. In these cases every effort should be made to adapt closely the soft tissue over the socket margins. This procedure often necessitates the further removal of the buccal and many times the lingual plate in order to obtain sufficient relaxation of the tissue so that the defect can be closed. An attempt should be made to stimulate bleeding in the socket by lightly irritating the margins of soft tissue with a curette. The area then should be covered with a packet of sterile gauze, having the patient bite firmly on this dressing for ten to fifteen minutes. The patient should be instructed further to refrain if possible, from blowing his nose, sniffling, or sneezing.

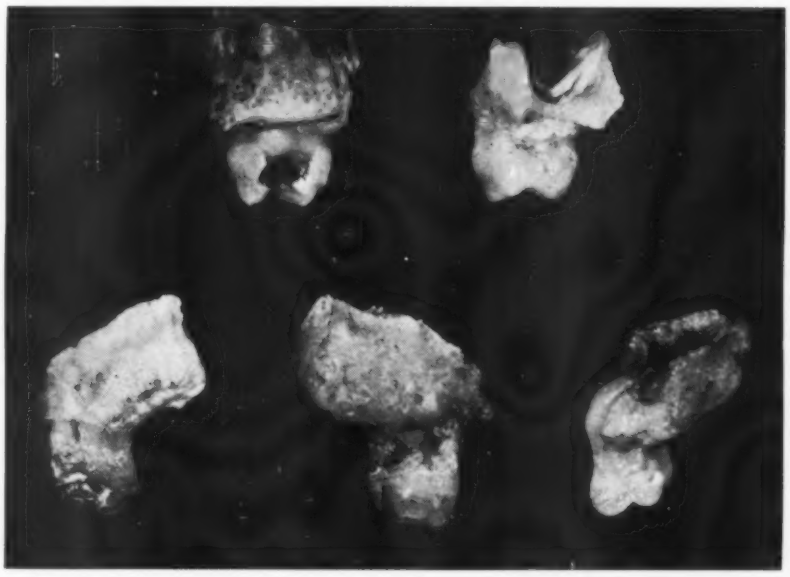


Fig. 6.—Specimen of fractured tuberosities which occurred during dental extraction.

MANDIBULAR MOLAR EXTRACTIONS

The removal of mandibular molar teeth can be simplified wherever necessary by applying the same principles and techniques that are used in the removal of maxillary molars.

SUMMARY

Extensive contributions have been made to dental literature concerning the management and technical procedures for the removal of difficult maxillary and mandibular third molar teeth.

It is advocated, where difficult oral surgical procedures are to be expected, that such cases be referred to the surgeon, whose experience and training make him better able to meet complications when they occur.

The recognition of difficult oral surgical problems demands a careful preoperative study and treatment plan. Otherwise, a procedure may be started without anticipation of the complications which may ensue. Such complications may disrupt a smoothly running operative program and tend to stimulate a more rapid surgical technique. In addition, increased physical and mental tension are created when working under such conditions, a situation which is not conducive to the best service for the patient.

There appears to be no need for the discussion of the removal of difficult first and second molars. The removal of these particular teeth presents very real problems of management to the operator.

The importance of diagnosis, careful preoperative radiographic examination, the observation of asepsis, and the procurement of profound anesthesia are stressed.

Adequate accessibility to the operative field is an important surgical principle which should always be obtained.

The use and abuse of incisions is discussed, with the conclusion that incisions rarely are necessary in order to gain satisfactory visibility.

Observing these basic surgical principles, various technical procedures are presented which will aid and simplify the removal of difficult molar teeth.

THE MECHANICS OF THE TEMPOROMANDIBULAR JOINT: ILLUSTRATED BY TWO CASES

ROBERT W. ROOT, B.S., D.D.S., M.S.

THE temporomandibular joint is a ginglymo-arthrodial joint, i.e., it combines the functions of a hinge joint and a gliding joint. Situated between the mandibular fossa and the condyle is a highly specialized structure, the articular disc. This is an oval fibrocartilaginous plate; its upper surface may be made to conform to the mandibular fossa and the articular eminence, thus making it concavoconvex from the anterior to the posterior relationship, whereas the lower surface fitting over the condyle is concave. This structure divides the joint into two cavities, the meniscotemporal and the meniscocondylar, each of which has its own synovial membrane. In some cases, however, the disc is perforated and the cavities communicate with each other.

The periphery of the disc is attached to the loose articular capsule, except in the front portion to which the sphenomeniscus head (or part) of the external pterygoid muscle is attached. The joint is enclosed by this thin capsule, which is attached above to the circumference of the mandibular fossa and immediately in front of the articular tubercle, and below to the neck of the mandibular condyle. Supporting this capsule are several ligaments, one of which is the temporomandibular ligament covering the outer aspect of the capsule. It consists of two bands, one in front of the other, attached above to the lateral surface of the zygomatic arch and to the tubercle of its root on its lower border, and below to the lateral surface and posterior border of the neck of the condyle. The ligament is broader at its superior attachment and the fibers are directed obliquely downward and backward. The inner surface of the capsule is not as well supported as the outer surface. The sphenomandibular ligament is attached above to the spine of the angle of the sphenoid and below along the posterior border of the ascending ramus from the neck of the condyle to the level of the lingula of the mandibular foramen, attaching anteriorly on the medial surface of the ascending ramus at this same level. The external pterygoid lies in contact with this ligament on the lateral surface above; below the ligament is separated from the neck of the condyle by the internal maxillary vessels, and, still lower, the inferior alveolar vessels and nerve. A lobule of the parotid gland lies between it and the ramus of the mandible. The stylomandibular ligament extends from the styloid process of the temporal bone to the angle and posterior border of the ramus of the mandible as far as the neck of the condyle. These last two ligaments have no direct connection with the capsule of the joint but rightfully should be considered as ligaments of the temporomandibular joint, because of the important part they play in the movements of the mandible.

The complicated mechanism of the temporomandibular articulation overshadows that of any other joint in the human anatomy. Not only does it have in reality two joints, the meniscotemporal and meniscocondylar articulations, but with every action in the one joint there must be a similar or compensating action in the joint of the opposite side, thus adding to the complexity and difficulty in understanding this articulation. To further add to the problem is the influence the articulating teeth must have on the functioning of the joints.

The question arises as to how the temporomandibular joint functions. There are numerous concepts and theories concerning the movements of the lower jaw. Dr. Frederick Lord[1] in his article has given a very excellent and adequate discussion of these movements; although differing from many anatomy texts in their explanations he has gained a great many followers in his logical presentation of this subject. He and others have proved that the head of the mandible always moves forward with every movement in opening the mouth.

The sphenomandibular and stylomandibular ligaments are attached to the ramus at the level of the mandibular foramen, which is the entrance of the vessels and nerves to the mandible. In nearly all the moving parts of the body, vessels and nerves enter their designated structures at the point of least motion. This fits in with the accepted fact that the transverse axis of rotation for the lower jaw is at the midpoint of the ascending ramus.

The articular disc compensates for the discrepancies of the fitting of the condyle into the glenoid fossa of the temporal bone. In doing so, it varies greatly as to its thickness in that portion which lies directly over the condyle when the jaw is closed, while it is thinnest in the area lying between the lowest point of the articular eminence to the highest point of the glenoid fossa. The arrangement of the disc in regard to the thick and the thin portions is of the greatest importance in the mechanism of this articulation. As the condyle moves forward in the opening of the mouth to take its normal position directly beneath the eminence, the articular disc must also move forward, bringing the thinnest portion of the disc directly beneath the eminence; otherwise, the condyle would meet heavy resistance due to the thickness of the disc in that area and would be unable to advance smoothly and in a straight line from the closed to the open position of the jaw.

It is universally accepted that the closure of the jaw is brought about by the joint action of the temporal, the masseter, and the internal pterygoid muscles. They can all pull upward and forward, forcing the teeth together. The attachments of these muscles fit the pattern for all muscles for the reason that the power arm is shorter than the weight arm, a fact that applies to the external pterygoid muscle as a proof of its function to open the mouth.

Trituration is carried out by the contraction of the external pterygoid muscle on one side and not on the opposite side, thus causing the mandible to shift or swing to one side.

The "natural" opening of the mouth, as in eating, is affected by the action of the external pterygoid muscle. The superior fibers of the upper head of the

muscle which is less powerful than the lower head are attached to the front edge of the meniscus and pulls the disc forward. The lower head, which is powerful, is attached to the neck of the condyle and pulls the condyle forward. As these muscles contract, the mandibular head slides forward to a position beneath the articular tubercle and the sphenomandibular and stylomandibular ligaments become taut, prohibiting further forward movement of the midramus region. Further contraction causes the mandible to rotate on an axis through the region of insertion of these ligaments. (Fig. 1.) However, in "forced" opening of the mouth, the backward pull of the inframandibular muscles come into play by offsetting the forward pull of the external pterygoid muscle. The axis of rotation of the mandible is maintained at the midramus, and the backward pull of the inframandibular muscles prevents the two ligaments from being torn or stretched by a too powerful pull of the external pterygoid muscles.

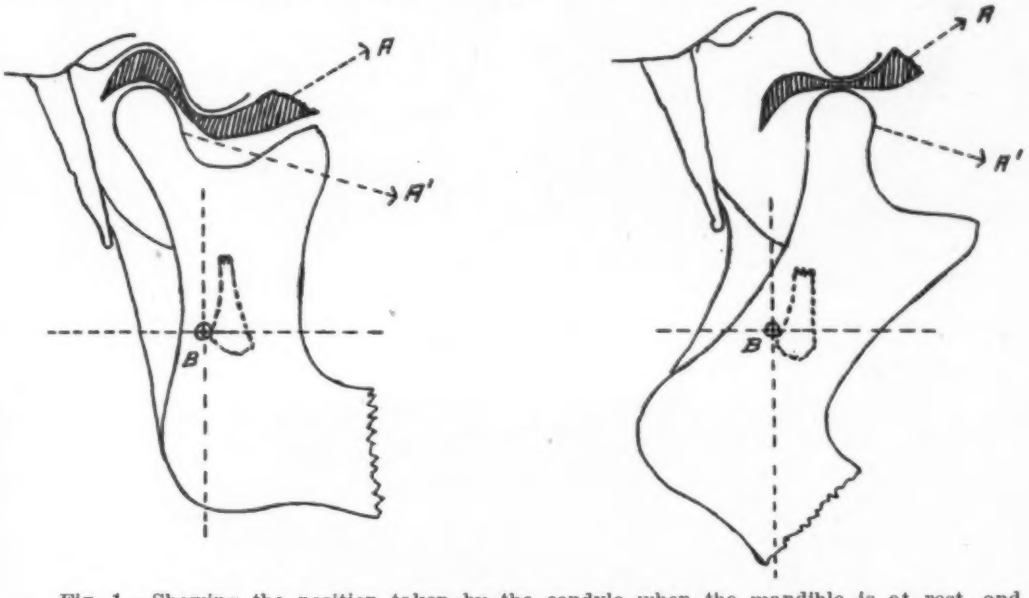
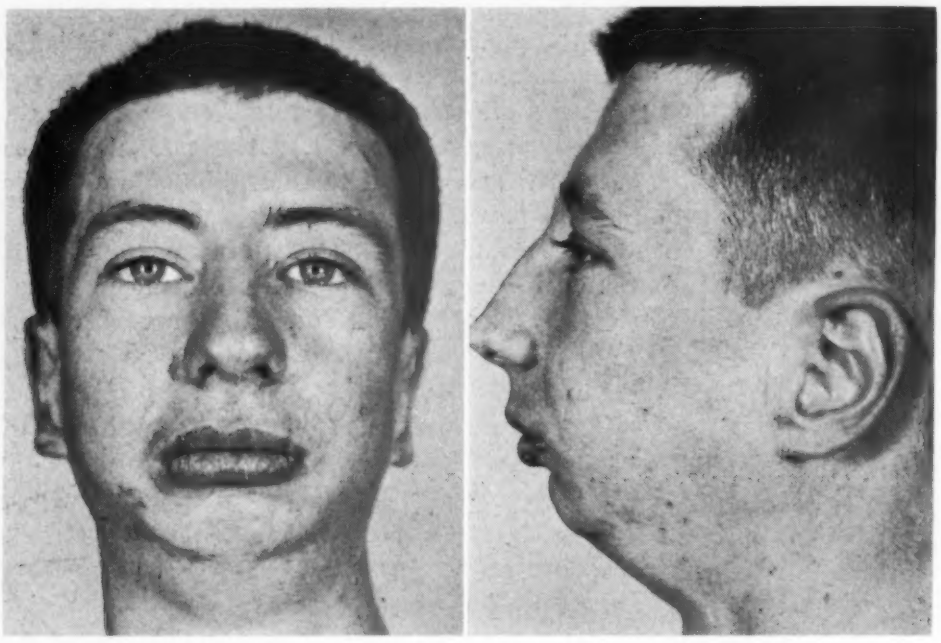


Fig. 1.—Showing the position taken by the condyle when the mandible is at rest, and following contraction of the external pterygoid muscle. *A* and *A'* show direction of pull of the upper and lower heads respectively. The axis of rotation, *B*, is maintained by the stylo- and sphenomandibular ligaments.

Interesting clinical observations regarding the opening of the mouth were made on two patients on the Oral Surgery Service at the University Hospital:

The first case was that of a bilateral osseous ankylosis of the temporomandibular joints. The patient, a man, 19 years of age, presented the characteristic facial deformity and oral condition seen in this type of case where the onset had taken place in early childhood (Figs. 2, 3, and 4). At 7 years of age the patient developed a bilateral mastoiditis following an attack of scarlet fever and diphtheria. A bilateral mastoidectomy was done, and approximately one year later it was noticed that he had increasing difficulty in opening his mouth. This steadily increased up to the time he was 12 years of age when he was unable to separate his teeth at all. An operation was performed at that time in an attempt to release the temporomandibular articulation. According to the patient's mother, following the operation he was still unable to open his mouth. Roentgenograms of the temporomandibular joints showed apparent complete ankylosis of the left temporomandibular joint, and partial ankylosis and pronounced irregularity of the right temporomandibular joint (Figs. 5 and 6). A bilateral mandibular condylectomy was done in which approximately the upper third of the posterior

portion of the ascending rami were removed, leaving the coronoid processes intact (Figs. 7 and 8). This was done in two operations, the right side first, and the opposite side eighteen days later. Immediately following the second operation he was able to open his mouth wide.



Figs. 2 and 3.—Photographs showing underdevelopment of mandible in bilateral ankylosis of the temporomandibular joint.

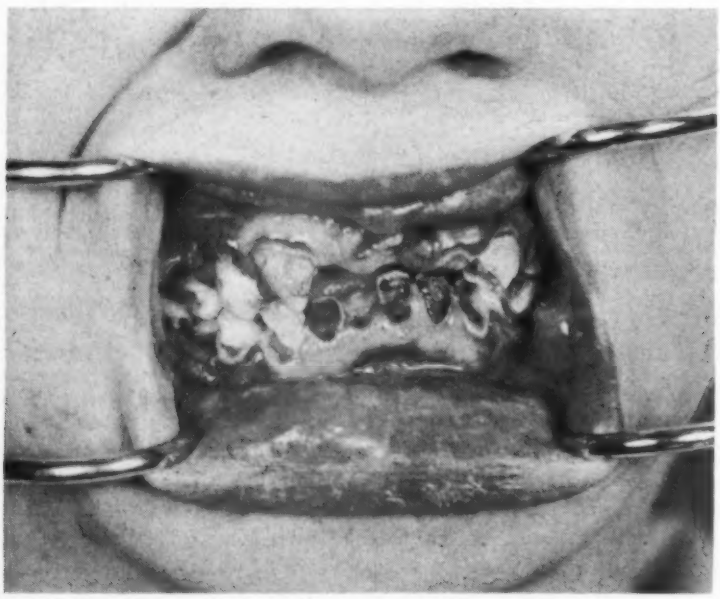


Fig. 4.—Photograph showing oral sepsis, retained roots, and extensive caries.

Following the operations there was definite visible and palpable evidence of marked contraction of the inframandibular muscles upon opening the mouth, which is not observed in the normal individual except in "forced" opening.

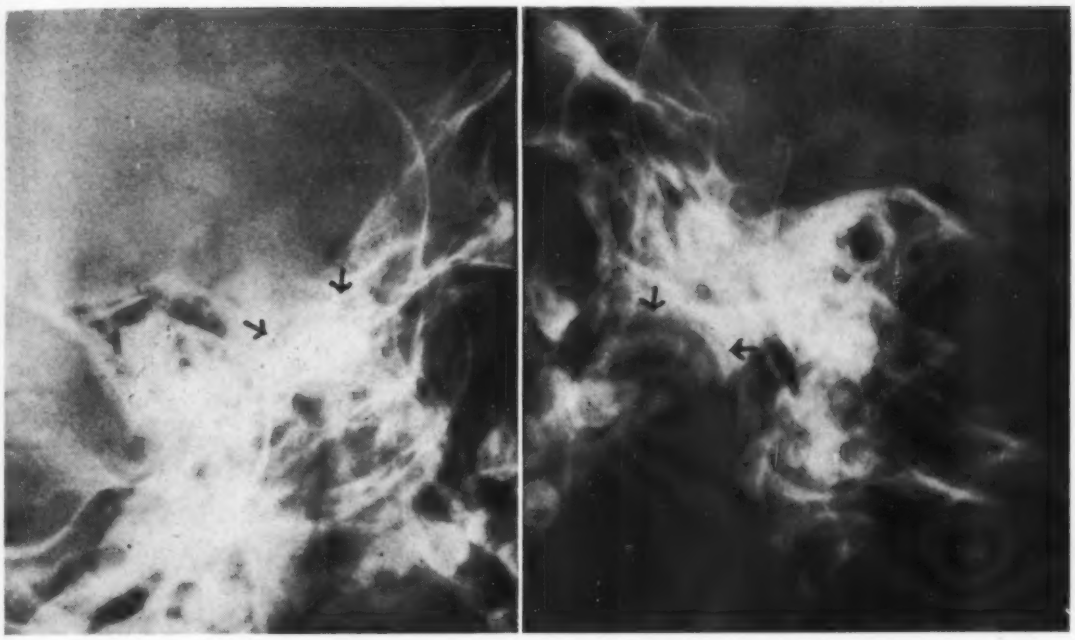
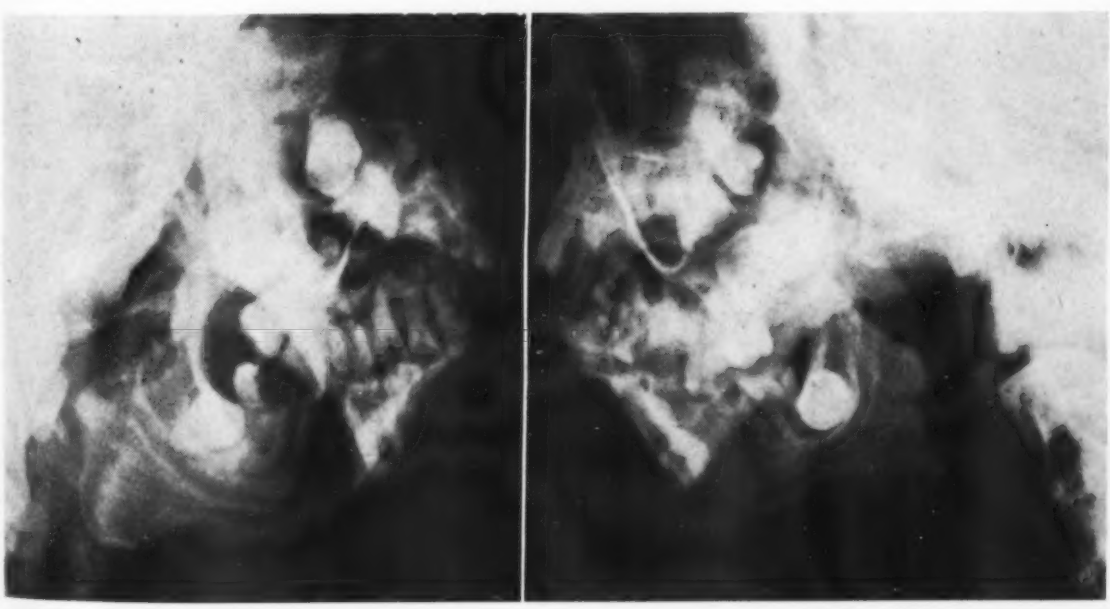


Fig. 5. Fig. 6.

Fig. 5.—Roentgenogram of the left temporomandibular joint.
Fig. 6.—Roentgenogram of the right temporomandibular joint.



Left Right

Figs. 7 and 8.—Postoperative roentgenograms showing the portion of the ascending rami removed.

The second case was that of a severe gunshot wound to the face. The patient, a man, 33 years of age, sustained a self-inflicted shotgun wound to the inferior portion of his face in which the entire floor of the mouth as well as the anterior portion of the mandible was carried away, destroying all the attachments of the inframandibular muscles, namely, anterior



Figs. 9 and 10.—Postoperative photographs showing correction of facial deformity by cartilage implant to chin.

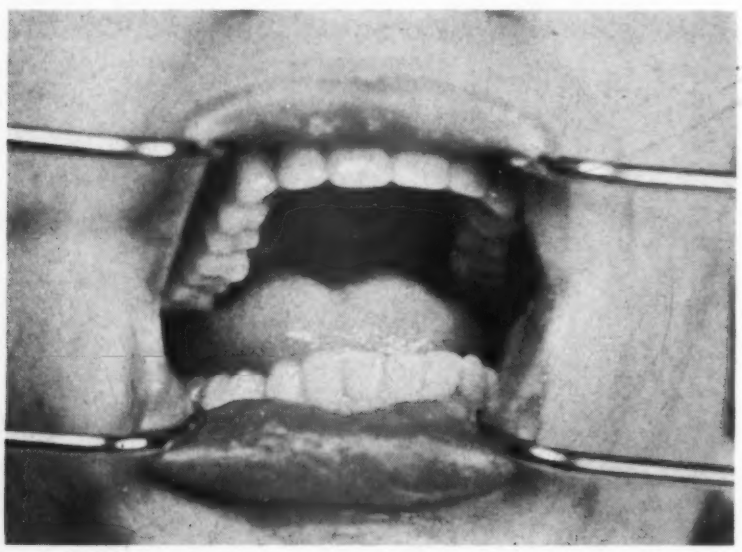


Fig. 11.—Photograph following extraction of teeth and construction of dentures.

belly of the digastric, geniohyoid, mylohyoid, and platysma muscles (Fig. 12). The patient was able to depress that portion of the right body of the mandible which remained intact in spite of the fact that the inframandibular muscles were no longer functioning. The external pterygoid muscle, other than those muscles which function in closing the mouth, was the only muscle remaining attached to function in the opening of the mouth. He was able to do this without the aid of the inframandibular muscles.



Fig. 12.—Photograph showing extent of destruction of the inframandibular structures from gunshot wound.

CONCLUSIONS

A short discussion of the anatomy and mechanics of the temporomandibular joint is given.

Two cases are presented which demonstrate clinically the muscles which take part in opening of the mouth.

The external pterygoid muscle is the muscle of mastication that takes the major role in the "natural" opening of the mouth. It is able to do this in the complete absence of the other muscles accredited with the function of opening the mouth.

With the loss of the function of the external pterygoid, the inframandibular muscles may compensate for this loss by producing a "forced" opening of the mouth.

REFERENCE

1. Lord, Frederick P.: Movements of the Jaw and How They Are Effected, INT. J. ORTHODONTIA 23: 557, 1937.

ANKYLOSIS OF THE TEMPOROMANDIBULAR JOINT

REED O. DINGMAN, D.D.S., M.D.*

CHRONIC fibrous or fibro-osseous ankylosis of the temporomandibular joint is a disease requiring surgical treatment for its correction. In 1851 Esmarch[4] first attempted to relieve a patient with ankylosis of the temporomandibular joint by surgical intervention. He removed a wedge-shaped section of bone from the body of the mandible, but the operation was unsuccessful. Other methods were proposed by various surgeons who made valuable contributions to our knowledge of this condition, but it was not until the work of Murphey[8] and Blair[1] that the procedure was standardized and the results generally satisfactory.

Ankylosis of the temporomandibular joint is a serious condition because it interferes with mastication and nutrition, prevents proper oral hygiene, precludes the possibility of adequate dental care, and inhibits the functional motion that in children stimulates mandibular growth. Proper enunciation is impossible. Inability to open the mouth might also lead to serious consequences in the event of the development of acute surgical disease of the oral cavity, pharynx, esophagus, or air passages.

Although not a common disease, the condition is frequently seen in an active surgical service. During the past fifteen years we have seen 38 cases of true ankylosis in the University of Michigan Hospital clinics. Males are more often afflicted than females only because of greater exposure to hazardous situations. The onset may be at any age, but usually the condition is seen in the younger age groups. Unilateral involvement is most often seen, but both joints may be involved.

Ankylosis of the temporomandibular joint may be either extra-articular, i.e., due to pathology not directly affecting the joint, or intra-articular, in which there is disease of the joint structures and surfaces. The extra-articular or false ankylosis is caused by limiting scar-tissue contractures of skin, mucosa, or muscle structures attached to the mandible or in the vicinity of the joint. The formation of scar in these tissues may follow mechanical, bacterial, thermal, or irradiation trauma. Osseous tumors of adjacent bony structures and depressed fractures of the zygomatic arch may impinge upon the ramus of the mandible and limit motion of the joint.

This discussion is limited to the true or intra-articular type of ankylosis, the chief etiological factors of which are trauma and infection. Foman[6] states, "Chronic temporomandibular ankylosis may be either congenital or acquired." Although theoretically possible, the condition is probably rarely

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if ever a congenital finding. It is seen occasionally very early in life but is undoubtedly due to trauma suffered at the time of passage through the birth canal or to injury by forceps delivery.

The most common etiological factor is mechanical trauma to the mandible. The force may or may not result in fracture or dislocation, but sufficient injury is produced to result in progressive destructive changes within the joint cavity, which finally leads to complete fixation. One of our cases of bilateral ankylosis developed following healing of bilateral unreduced fractures of the condyle neck with lateral dislocation. Another developed as the result of meddlesome surgery in an attempted open reduction for fracture of the condyle neck. Other causes are: chronic osteo-arthritis; suppurative processes of the middle ear, parotid gland, skull, or mandible that extend into the joint; and metastatic infectious processes of the joint, sometimes seen during the course of acute childhood exanthematous diseases, scarlet fever, and acute gonorrhea. In one of our cases congenital syphilis was the only cause that could be determined.[2]

The pathologic findings, regardless of the etiological factors operating, are usually the same in all cases of intra-articular or true ankylosis. The limiting tissue may be either fibrous, osseous, or both, the difference undoubtedly being simply a matter of degree of development of the disease. The process is progressive and consists of destruction of the articular surfaces, meniscus, capsule, and adjacent ligaments immediately supporting the joint by fibrotic changes with subsequent osteoblastic activity and ossification, resulting in complete obliteration of the joint space. In the advanced condition the condyle head becomes continuous with the temporal bone. There is often marked thickening of the condyle head and neck and in some instances complete obliteration of the sigmoid fossa of the mandible by a mass of bone fused to the coronoid process of the mandible and to the zygomatic arch.

The diagnosis of temporomandibular ankylosis is made on the history of long-standing inability to open the mouth following injury or an inflammatory process in the joint. The condition is of a chronic nature and generally is not difficult to differentiate from the acute and transitory limitations of motion seen in local reflex spasm, hysteria, tetanus, parotitis, and acute inflammatory reactions of the joint or muscles of mastication. Ankylosis developing during the developmental period of the mandible results in retardation of growth and deformity on the affected side. In unilateral involvement the normal side develops eccentrically toward the affected side, giving flatness of the lower part of the face on the uninvolved side. This may lead to confusion in diagnosis and we have seen patients who have had surgery done on the normal joint. As pointed out by Blair,[1] a very useful diagnostic point is an unusually prominent angle of the mandible with accentuation of the notch at the lower border just in front of the angle. This finding is present only in the case developing during childhood.

In unilateral cases some motion can usually be detected in the normal joint on palpation and the symphysis shifts slightly toward the involved side on attempting to open the mouth.

In bilateral joint involvement of childhood, the retardation of growth of the mandible is symmetrical and gives rise to a bird-like profile characteristic of micrognathia. The deformity is accentuated by protrusion of the maxilla and anterior maxillary teeth as a result of forcing food between the teeth with the thumb or spoon handle.

The degree of deformity is inversely proportional to the age of onset of the process. The bilaterally ankylosed joints permit no motion. The apparent motion in some cases is due to the inherent elasticity of the bone.

Hygiene is difficult because of inability to open the mouth and the crowded condition of the teeth in the ankylosed jaw. Oral sepsis, peridontoclasia, and advanced caries are the usual findings.

In ankylosis developing after mandibular growth has been attained, deformity is not a finding except as it is incident to dislocation or fracture of the mandible.

Adequate x-rays of the joint carefully taken to prevent superimposition of other structures are very useful in demonstrating the degree of destruction of the joint. Anteroposterior and semilateral views should be taken using stereoscopic technique.

Treatment is so uniformly successful that all cases should be subjected to surgery. Surgical intervention is indicated because of the improvement of function it offers and also because of the psychological effect upon the patient. Many patients are more concerned about the appearance of the deformity and the embarrassment of eating in public than about the functional disability. The younger patients especially are liable to develop personality complexes, inferiority feelings, or become behavior problems because of embarrassment or because of ridicule by their associates.

Dilators, exercisors, or instruments for forceful opening of the mouth are ineffective and useless. The only satisfactory treatment is surgical approach to the joint and excision of the ankylosed bone.

Whenever possible, the operation should be done under local anesthesia because of the greater safety factor incident to its use. The properly premedicated patient suffers neither physical nor psychic trauma when operated upon under local anesthesia.

Block or infiltration techniques or both are satisfactory and offer the additional advantage of hemostasis when 25 to 30 drops of adrenalin per ounce are added to the novocain. Very young and uncooperative patients do better under general inhalation anesthesia administered by nasopharyngeal tube.

The hair is shaved from the preauricular region to the level of the free upper margin of the helix. The usual skin preparation is carried out and the ear canal lightly packed with sterile cotton. Various incisions are advocated but we find the auricular incision desirable because it permits adequate exposure and minimizes the postoperative scar. (Fig. 1.)

The incision is started in the fold at the junction of the anterior margin of the helix and the temporal skin just below the level of the upper attachment of the ear. It is carried downward in this fold to the upper portion of

the tragus where it is continued just inside the margin of the tragus to the anterior fold of the lobule. At this point it again becomes visible and is carried down to the lower attachment of the lobule. The incision is carried through the skin and immediate subcutaneous tissues, and the flap is dissected anteriorly for about 2½ centimeters. The flap is then folded forward upon itself where it is sutured out of the way with a stitch to the skin of the face.

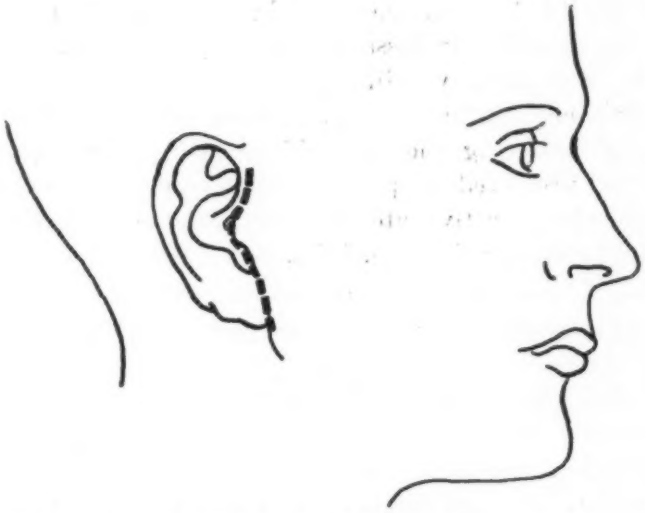


Fig. 1.—The skin incision is designed to give adequate exposure with a minimum of post-operative disability.

The incision through the subcutaneous fascia is carried down to the temporal and masseteric fascia but is made slightly anterior to the plane of the skin incision in order to avoid the cartilaginous portion of the external auditory canal. The upper part of the incision can be made with safety with regard to the seventh nerve. The lower part of the incision must be made with care because of the close proximity to the main branches of the nerve. In the average adult patient the limit of safety is 2 cm. below the upper level of the zygoma. According to recent studies of surgical anatomy of the facial nerve by McCormack, Cauldwell, and Anson[7] "the point of bifurcation of the facial nerve lies posterior and slightly medial to the ramus of the mandible and superiorly two-thirds of the distance between the external angle and the palpable condyloid process (temporomandibular articulation)." This flap is undermined along the plane of the temporomasseteric fascia and carries with it the temporal vessels and the upper portion of the parotid gland. It is retracted forward where it is held with stout small retractors. The fascia and periosteum is incised over the zygoma and along the posterior aspect of the joint. The posterior part of the masseter muscle is detached from the zygoma by blunt dissection and is retracted downward and forward to expose the joint. The tissues along the posterior aspects of the condyle neck are retracted backward. The head of the condyle is excised by means of chisels and bone forceps. A bone drill may be used to cut across the condyle neck if desired but this

should be done only when the tissue medial to the area being cut is protected by a curved flat instrument. This prevents damage to the internal maxillary artery, third branch of the fifth nerve, and large veins that lie medial to the condyle neck.

It is desirable to have at least a 2 cm. separation between the cut margin of the mandible and the temporal bone in order to prevent recurrence. This amount of bone separation is usually not difficult to attain in the adult if the condyle is cut at the level of the sigmoid fossa and the bone is carefully chiseled from the mandibular fossa of the temporal bone. The procedure is more difficult in cases of bony obliteration of the sigmoid fossa with fusion to the zygoma. This necessitates removal of bone across the entire width of the ramus, sometimes including the coronoid process. The temporal muscle insertion should be preserved if possible. Inadequate bone removal with regeneration across the operative site has been the cause of failure in the recurrent cases of ankylosis seen in our clinics.

Interposition of pedicle flaps of temporal muscle and fascia, as well as free fascial, fat, and muscle transplants have been described. Figi[5] inserted a cartilage block in one case to provide an articulating surface with good results. The use of foreign substances such as metals, acrylics, or heterografts should never be considered under any circumstances. Since the ultimate fate of muscle, fat, or fascia insertion is atrophy with replacement by fibrous tissue, it seems unnecessary to complicate the procedure with these grafts as the same final result can be attained by allowing the cavity to fill with a blood clot and organize into fibrous tissue. We have proved to our complete satisfaction that it is unnecessary to insert any type of material into the operative defect.[3]

The wound is closed carefully in layers with fine plain catgut for the fascia and silk or dermalon for skin approximation. A small sterile rubber band is inserted into the depth of the defect to act as a drain. One end is permitted to protrude through the lower end of the incision and is removed in forty-eight hours.

It is not necessary to open the mouth forcibly or to block the mouth open during the course of the operation or postoperatively. The mouth will gradually open and function very well in a short time without any exercise other than that of mastication. Chewing gum may hasten the recovery.

In one of our bilateral cases operated upon in one stage, a retrusion deformity of about 4 mm. resulted. This was not severe but was sufficient to throw the teeth out of normal occlusion. In subsequent cases it has been found desirable to operate the bilateral ankylosed joints in two stages allowing from four to six weeks to elapse before operation on the second side. This lapse of time permits sufficient organization on the first side operated upon to prevent posterior displacements of the mandible. Function in bilaterally operated patients is excellent except for protrusive and lateral motion, both of which are not possible because of destruction of the insertion of the external pterygoid muscles. The verticle dimension is maintained by the occlusal relationships.

SUMMARY

Chronic ankylosis of the temporomandibular joint is discussed from the standpoint of incidence, etiology, pathology, diagnosis, and treatment. Complete excision of the condyle head under local anesthesia, whenever possible, is the preferable method of treatment. An auricular incision provides adequate access to the joint and gives the best cosmetic results. Bilateral cases should be done in two operative procedures with an interval of at least one month between operations.

CONCLUSIONS

1. Ankylosis of the temporomandibular joint is a surgical disease.

2. Forceful opening of the jaws has no place in the treatment either before or after operation.

3. The auricular incision is preferable from the standpoint of safety to the branches of the seventh nerve and because the cosmetic result is more pleasing.

4. Adequate bone excision is imperative in attaining a permanent functional result.

5. Interposition of any material between the cut bone surfaces is unnecessary.

6. Bilateral ankylosis of the temporomandibular joint should not be operated upon at one sitting.

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FOCAL INFECTION

A Review of the Recent Literature

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THERE is much in recent publications to show the change of opinion regarding the relationship of focal infection to systemic disease. This paper will review the literature as it applies to the theory of focal infection in rheumatoid arthritis and iritis, with special attention to the part dental infection may play.

Rosenow's[1] work has probably served as the basis for exploitation of the theory of focal infection. He states that "the causative streptococcus often was isolated not only from badly infected tonsils, but from those that seemed normal but which, on removal, were found to be large and sometimes to contain abscesses. The causative streptococcus also was obtained from unsuspected remnants of tonsils and from symptomless roentgenographically negative pulpless teeth root tips, residual regions in the jaw which had not been considered previously or which were thought not to be harmless." Rosenow never seems to have taken into account the fact that it would be almost impossible not to recover streptococci from the regions which he cultured, using the methods he used. He made no attempt to rule out contamination by green-producing streptococci normally found in these areas. Sommer and Crowley[2] found that when intact teeth were cultured via the root canal using an aseptic technique, bacteriologic findings did not wholly confirm the roentgenographic appearance of periapical lesions. Too much emphasis should not be placed on the roentgenogram as a diagnostic agent in condemning questionable teeth; negative cultures were obtained from all types of roentgenographic lesions.

Rosenow's reports led to a great deal of work by internists, bacteriologists, and pathologists, who after much effort found they could come to no definite conclusions as to the bacteriologic relationship of focal infection to rheumatism. This is best exemplified by the work of Cecil and Archer,[3] who in 1927 published an article stating that, as a result of a study of 200 cases of rheumatism, definite relationship could be traced to foci of infection such as teeth, tonsils, and sinuses; that a large number of cases had improved after removal or treatment of such foci. However, in 1938, Cecil with Angevine[4] reported another such series of cases in which they contradicted their original findings. They pointed out that many of the cases reported in the original study would no longer be classified as suffering from rheumatoid arthritis because of the greater knowledge of the differential diagnosis of the disease. Many of the

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patients reported cured or improved, after longer study had recurrences of the disease. They reported, too, that they saw many patients with no known focus of infection who still had rheumatoid arthritis.

Reimann and Havens[5] in 1940 published an article which thoroughly reviews the literature on focal infection. They point out that the early bacteriology on which this theory is based is not acceptable today. They quote the work of Tunnicliffe and Hammond to show that cultures cannot be relied upon to show either absence or presence of infection in extracted teeth, since the possibility of contamination by bacteria in the mouth (of which *Streptococcus viridans* is one of the predominating organisms) cannot be ruled out. The mere presence of bacteria does not mean infection. Negative cultures may be obtained from extracted teeth if an effort is made to sterilize the outside of the tooth in order to culture the tooth contents. Kanner[6] in 1938 showed that the manipulation of the tooth in the process of extraction forced organisms into the pulp tissue.

Reimann and Havens' article discusses the relationship of focal infection not only to rheumatoid arthritis but to many other diseases which have been thought at one time or another to be related to foci of infection. They conclude: "(a) The theory of focal infection in the sense of the term used here has not been proved, (b) the infectious agents involved are not known. (c) patients whose teeth or tonsils are removed often continue to suffer from the original disease for which they were removed, (d) beneficial effects which occasionally occur after surgical measures are often outweighed by harmful effects or no effect at all."

Slocumb, et al.,[7] of the Mayo Clinic do not agree with Reimann and Havens' conclusions. They state that because bacteriologists have been extremely critical of proof obtained by clinical evidence alone and because bacteriologists have failed to draw any conclusions by exact scientific methods, they will avoid this phase and evaluate the concept of focal infection on clinical observations alone. However, they quote Hench as saying that he had come to two conclusions as to the microbic theory of the origin of chronic infectious arthritis: As a clinical investigator he concludes that the cause of atrophic arthritis is unknown and that the evidence for infection, while impressive, is incomplete. As a practicing physician he feels he cannot wait for this evidence to be completed. Therefore, as a practicing physician he is committed to the microbic theory.

In 1942 the "Primer on Arthritis" discusses the classification of rheumatism, its etiology (it is pointed out that in some types the etiology is known), pathology, symptomatology, differential diagnosis, and treatment. It is pointed out that a possible relationship exists between hemolytic streptococci of Lancefield's Group A and rheumatoid arthritis. They state that 40 to 50 per cent of the patients' sera will agglutinate this group.

Sommer and Crowley found, however, that only 2 per cent of infected teeth cultured under aseptic conditions harbor *Streptococcus hemolyticus.* This would seem to show that teeth could be suspected of being the original foci of infection in a very small number of cases.

Boland, Headley, and Hench[9] in 1944 published a report on the effect of penicillin on rheumatoid arthritis. Ten soldiers with early but progressive rheumatoid arthritis were treated with excessively large doses of penicillin for fourteen to twenty days. Their results "offer no support to the idea that hemolytic streptococci may be etiologically related to rheumatoid arthritis. In view of negative results with rather large doses of penicillin it does not seem unreasonable to assume that rheumatoid arthritis is not caused by any of the bacteria which are already known to be rapidly affected by penicillin."

Woods,[10] in 1942, published an excellent review of the literature for and against the possible etiological relation of focal infection to iritis. He mentions the fact that while with "bacteriologists, pathologists and internists the doctrine of focal infection stands on a shaky pedestal and clad in somewhat dilapidated raiment, among ophthalmologists the case is different"—it is an established procedure to search for foci of infection in the study of the etiology of uveal-tract disease.

The author points out that many cases of iritis and choroiditis of undetermined etiology recover spontaneously; also there are long remissions before recurrences in the optic nerve and elsewhere reveal the true nature of the disorder. A majority of reports attributing eye disturbances to foci of infection, according to the author, are open to criticism for (1) they do not give the complete material from which the cases reported are drawn (2) they emphasize some one factor believed to be of importance and do not give a comprehensive picture of possible etiology, (2) the data presented do not permit deductions concerning the relative importance of focal infection as compared to other possible etiological factors.

TABLE I (Table 5 in original article*). SUMMARY OF THE STUDIES OF IRONS AND BROWN, IRONS, AND GUYTON AND WOODS, ON THE ETIOLOGY OF UVEAL DISEASE

REPORT	SYPHILIS (PER CENT)	TUBERCULOSIS (PER CENT)	FOCAL INFECTION (PER CENT)	GONORRHEA (PER CENT)	NONGRANULOMATOUS SYSTEMIC DISEASE (PER CENT)	MISCELLANEOUS AND UNDETERMINED (PER CENT)
Irons and Brown (1916 and 1923)	19	4	72			5
Irons (1931)	8.9(?)	15-56	24-64			
Guyton and Woods (1940)	10.5	49.7	26.1	4.6	5.9	3.2

*Woods, A. C.: Focal Infection, Am. J. Ophth. 25: 1423, 1942.

He reports four papers which are not subject to the above criticisms. The first was published in 1916, the last in 1940. The difference in importance attributed to focal infection is striking (See Table I). It illustrates the change of opinion in the course of two decades on the relative importance of tuberculosis and foci of infection as causes of uveal disease and the decline of the implicit belief in the doctrine of focal infection.

Against the argument that many cases of uveal disease are encountered for which no systemic cause can be found other than foci of infection, he shows

the following table from a study by Hughes (See Table II). This table shows that patients with cataracts, a disease not caused by infection, have as many foci of infection as those suffering from uveitis, which is an infection.

TABLE II (Table 6 in original article*). INCIDENCE OF FOCI OF INFECTION IN 523 PATIENTS WITH UVEITIS AND 507 CATARACT PATIENTS WITHOUT UVEITIS (CORRECTED STATISTICALLY TO CONFORM WITH AGE DISTRIBUTION BY DECADES)

TYPE OF PATIENT	NO. OF PATIENTS	FOCI OF INFECTION	
		DENTAL INFECTION (PER CENT)	SINUS INFECTION (PER CENT)
Patients with uveitis	523	27.1	12.4
Patients with cataract	507	25.8	13.0

*Woods, A. C.: Focal Infection, Am. J. Ophth. 25: 1423, 1942.

"In recent years American ophthalmologists have realized more and more the importance of tuberculosis and brucellosis as causes of ocular disease and the difficulty in reaching an accurate diagnosis in either of these is well known. Many of the cases of uveal disease formerly attributed to focal infection are probably instances of ocular tuberculosis and the presence of infected tonsils, sinusitis or periapically infected teeth only incidental and inconsequential findings." (See Table III.)

TABLE III (Table 7 of original article*). EFFECT OF REMOVAL OF FOCI OF INFECTION ON RECURRENCES OF UVEITIS

TREATMENT OF FOCI OF INFECTION	NO. OF PATIENTS	AVERAGE PERIOD FOLLOWED (YEARS)	PREVIOUS ATTACKS (PER CENT)	RECURRENCES (PER CENT)
Totally removed	42	4.5	55	33
Partially removed	11	3.8	73	36
Not removed	21	4.2	67	29

*Woods, A. C.: Focal Infection, Am. J. Ophth. 25: 1423, 1942.

"However, it is assumed that there may be a cause and effect relationship between a primary focus of infection and ocular disease through such a mechanism as sensitization and intoxication of the diseased eye by bacterial products absorbed from the focus of infection; a fair experimental basis can be advanced for such an assumption, although clinical evidence is lacking.

"The routine removal of minor and symptomless foci of infection as a cure-all for endogenous ocular disease has no place in modern ophthalmology."

CONCLUSIONS

A review of the recent literature seems to show a swing away from the general acceptance of the theory of focal infection. However, it would be just as serious a mistake for the dentist not to eliminate dental infection in patients suffering from rheumatoid arthritis and iritis. This can be done by conservative methods in many cases. However, it must not be forgotten that many teeth cannot be treated conservatively by root canal treatment, because of the morphology of some canals.[11] The general resistance of the individual to infection must be taken into consideration, also, in deciding whether to treat or extract a tooth. In addition, this type of treatment is contraindicated in molars with extensive periapical involvement.

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CASES OF PATHOLOGIC INTEREST

DONALD A. KERR, A.B., D.D.S., M.S.

THE cases in this report were selected from our routine material because of some interesting clinical or pathologic feature. They have little that is new to add to our clinical and pathologic knowledge, but perhaps will refresh for us some of the clinical and pathologic possibilities.

The history in most of the cases is very brief, because material was sent to the laboratory from practitioners, and sometimes no history was available.

CASE 1.—Complex, composite odontoma.

A 38-year-old white woman complained of recurrent headaches and pains in the cervical portion of the neck for one year. The patient had no other complaint. History and physical findings were essentially negative. Roentgenographic examination revealed an area of increased density about the size of a walnut in the upper third molar region. The mass was removed under local anesthesia. Recovery was uneventful. For one year the patient has been free of headache and pain in the neck.

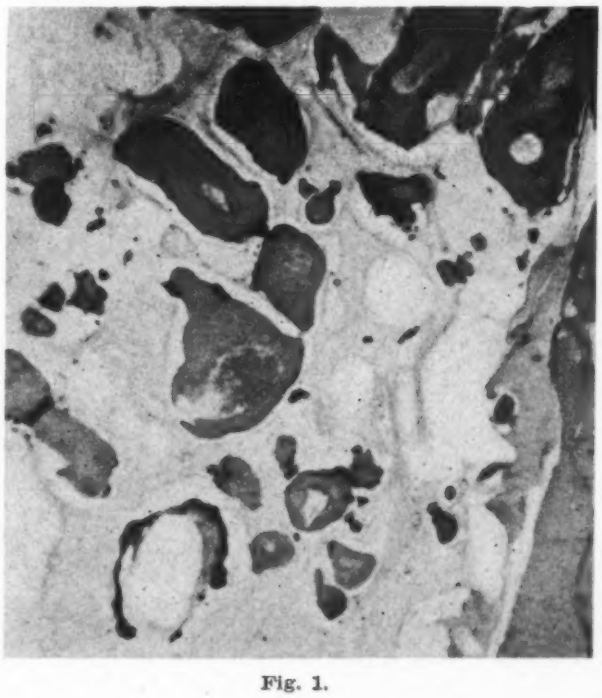


Fig. 1.

Fig. 1 demonstrates the character of the tumor removed. It was a complex composite odontoma of the type frequently seen in this region. It was composed of irregular masses of dentine surrounded by masses of enamel, enamel matrix, and some cementum.

Tumors of this type are fairly common in this region. This one is of interest because of the referred character of the symptoms and the complete relief of symptoms following removal.

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CASE 2.—Globulomaxillary cyst.

Two weeks previous to his appearance in the clinic, the patient noticed swelling and pain on the right side of the midline of the maxilla. The area was incised and drained by his dentist. The swelling was relieved, but the discomfort continued. Roentgenographic examination revealed a cyst above the right lateral incisor and cuspid. This extended to the maxillary sinus. A similar cyst was seen above the left lateral incisor. At operation, it was found that the cyst on the right side communicated with the maxillary sinus. On the left side there was no sinus involvement.

The cystic lining removed was composed of a thin epithelium of a columnar type, which appeared in most areas to be two cells in thickness and represented an attenuated respiratory epithelium. The epithelium covered a dense connective tissue containing numerous inflammatory infiltrations. The inflammatory process was much more severe in the membrane removed from the right side, but in all other respects the cystic membranes were essentially alike.

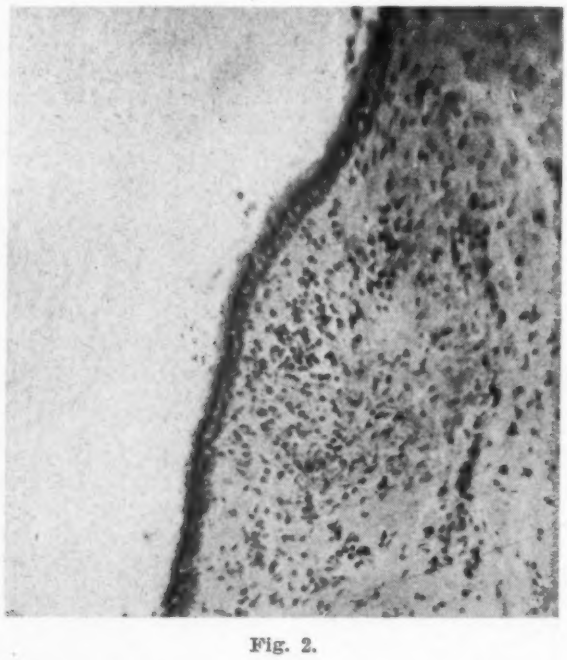


Fig. 2.

Fig. 2 shows the character of the cystic membrane removed from the left side.

These findings are characteristic of the globulomaxillary cysts, which are developmental inclusions at the line of union between the globular (median palatine) process and the maxillary process.

This case is reported to call our attention to the not infrequent bilateral character of this condition.

CASE 3.—Epuloid giant-cell tumor of the tongue.

A white man, aged 35 years, had a lesion on the right lateral aspect of the tongue of three months' duration. The lesion was pedunculated and not painful. Recently the lesion appeared to change in character and began to infiltrate the tongue. It produced some discomfort. Clinical examination revealed, at approximately the junction of the middle and posterior thirds of the tongue, on the right lateral aspect and extending to the dorsal and inferior surfaces, a firm, rubbery, ulcerated lesion, 3 cm. in diameter. There was no fixation of the tongue and the floor of the mouth was not invaded. All of the teeth on the right side were missing except the mandibular second molar which came in direct contact

with the lesion of the tongue. A biopsy of the lesion was reported "epuloid giant cell tumor of the tongue." Right hemiglossectomy was done.

The pathologic specimen revealed a lesion of the character and location described clinically. Microscopically, below the ulcerated surface was a marked fibroblastic and angio-



Fig. 3*A*.

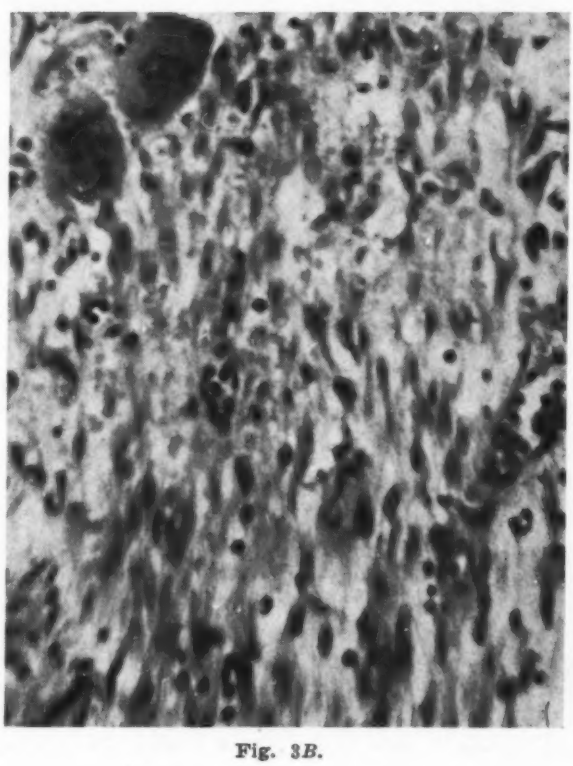


Fig. 3*B*.

blastic proliferation producing a very young immature connective tissue. The fibroblasts, although typical, were very large. The angioblastic proliferation formed many small, thin-walled, vascular channels. This zone of proliferation was very narrow and rapidly blended into an area which was characterized by the presence of numerous large multinucleated giant cells in an actively proliferating vascular stroma. The nuclei of the giant cells were of the same size and morphology as those of the proliferating fibroblasts, while the cytoplasm had a deeper staining quality. There appeared areas of transition from fibroblasts to giant cells. This resulted in a very cellular tissue which was invading and replacing the tongue muscle. In and around the areas of proliferation there was much old blood pigment in the form of hemosiderin. Many inflammatory cells of polymorphonuclear type were present beneath the area of ulceration.

Figs. 3*A* and 3*B* show the character of this pathologic change. It appears to be similar to the changes found in the giant-cell epulis associated with the jaws.

Treatment in this case was perhaps too radical, as this lesion is of local significance only. Recovery was uneventful and the patient has had no recurrence.



Fig. 4.

CASE 4.—Lymphangioma cysticum.

A white female infant, 17 months of age, was brought to the clinic because of an enlarged and bleeding tongue. The mother stated the child's tongue had always been large and had protruded from the mouth for the past seven months. Examination revealed a normal healthy child except for the enlarged protruding tongue which almost filled the oral opening. Its surface was dry and cracked and had some tendency to hemorrhage. There was a slight swelling of the submaxillary region. After injection with lipiodol, a multilocular cyst could be visualized in the floor of the mouth. A biopsy was taken, which showed the changes were those of a lymphangioma cysticum. By three separate surgical procedures, the tumors of the tongue and floor of the mouth were removed. The patient was discharged and appeared normal except for the small size of the tongue.

Congenital lymphangioma cysticum of the tongue, floor of the mouth and neck region, is not rare. When the tongue is involved extensively, there is very often an extension of the process into the neck region. This case is included because of the location of the dilated lymph spaces. Nearly all of the cystic areas were situated just beneath the epithelium. The entire process, although widespread, was located in the submucosa. There were large dilated lymphatic spaces in the connective tissue papillae which produced a thinning of the epithelium. In the deeper tissues, there were only a very few slightly dilated lymphatic spaces. This superficial terminal involvement is not the usual finding.

CASE 5.—Adenocarcinoma arising in mixed salivary gland tumor.

A 58-year-old white farmer presented himself with the chief complaints of swelling and discomfort in the floor of the mouth. The history revealed that a small nodule had been removed from the floor of the mouth by his dentist two years previously. The pathologic diagnosis was aberrant salivary gland. The mass then had recurred and gradually increased in size until there was a hard and movable mass present in the floor of the mouth on the left side, extending from the midline posteriorly for a distance of from 6 to 8 cm. Bimanual examination revealed a small egg-sized mass in the left side of the floor of the mouth. There was no fixation of the tongue. Physical examination was otherwise negative.

Microscopic sections of this tumor showed a marked variation in morphology. A large portion of the mass was composed of epithelial cells, which in some areas formed rather regular glandlike spaces of varying size. The cells were large with a vesicular nucleus and a rather intense-staining abundant pink cytoplasm. The boundary of the glandlike spaces was usually 2 to 3 cells in thickness. The spaces contained a mucinous substance. Near the capsule the pseudoglands were small and showed evidence of infiltration. In other areas, the epithelial cells were in small masses and cords. The masses were surrounded by a mucinous material. Where arranged in cords, the groups of cells resembled slightly the zona medullaris of the adrenal gland. Some of these characteristics are shown in Fig. 5*A*. In still another area the cells became very atypical and assumed a pleomorphic spindle shape. The cells were compactly arranged and had a slight amount of mucinous material associated with them. This character is shown in Fig. 5*B*.

This case is of interest for two reasons. First, it represents the rather rare pseudoadenomatous type of mixed salivary gland tumor so named because of the pseudoglands formed, and also because of its close resemblance to the pseudoadenomatous basal-cell carcinoma. Second, this neoplasm is of interest because of its malignant character, which is expressed by the ability to infiltrate, and marked atypical and cellular character in some areas. Adenocarcinoma arising in mixed salivary gland tumors are not a common finding.

CASE 6.—Papillary cystadenoma lymphomatosum.

Man, 51 years of age. "Has had this gland for 8 years—cervical gland." No further history was obtainable.

The mass grossly looked like enlarged lymph nodes. On section, they were found to be cystic, the cystic spaces filled with papillae. There was a small amount of serous fluid present. Throughout the papillae were whitish nodules.

Microscopically there was much lymphoid tissue present, arranged in branched papillae and masses with few germinal centers. The papillae were covered with a stratified or pseudostratified columnar epithelium. In some areas, it had a cuticular border. There was no evidence of mucin formation in the epithelial layer. Between the papillae was a coarsely granular albuminous precipitate containing a few leucocytes and desquamated cells. The remainder of the stroma consisted of lymphoid tissue, a delicate stroma, packed with lymphocytes, some showing germinal centers. The growth was typical and regular, stratified ciliated columnar epithelium upon a lymphadenoid submucosa, without any evidence of infiltration. This resembled very much the branchial cyst except for the character of the epithelium. (Fig. 6.)

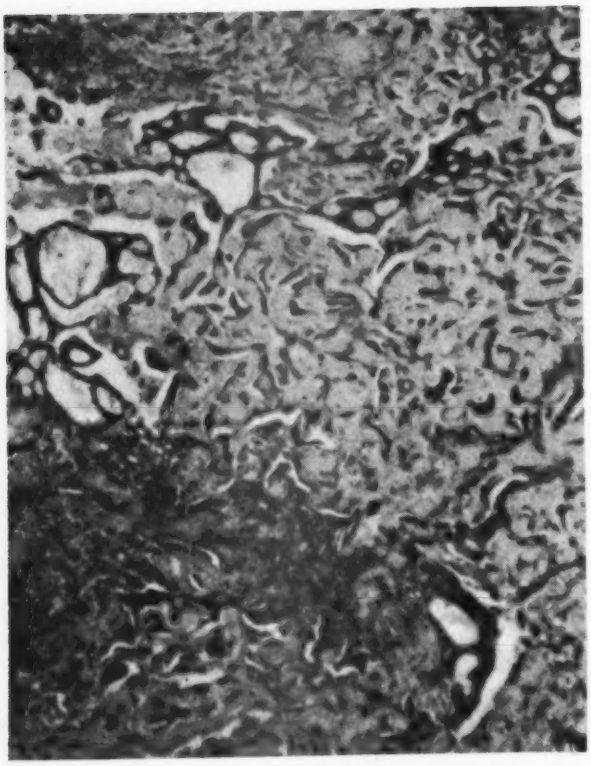


Fig. 5*A*.

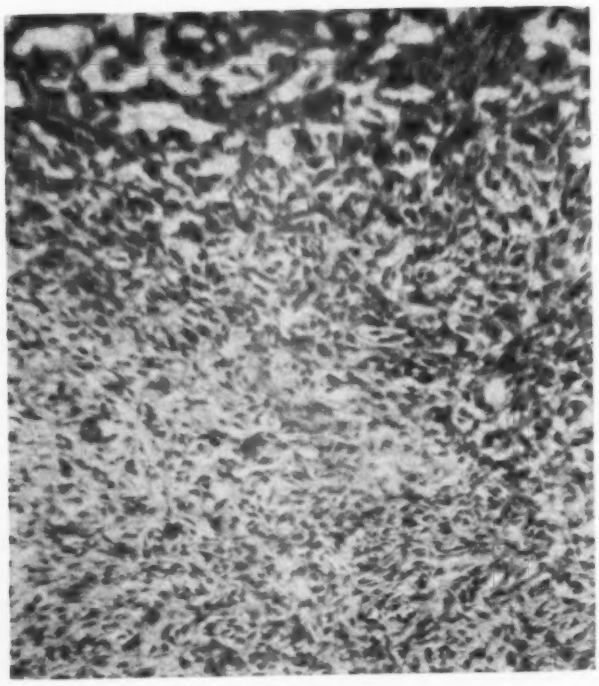


Fig. 5*B*.

This was a rare tumor found characteristically in the submaxillary and parotid area. Warthin[1] reported two cases in 1929. There were two out of several thousand tissues examined, of which over 700 were mixed salivary gland tumors, and over 500 were branchial cysts of the cervical region. In 1929 there were almost no reports in the literature. At present there are several reports, but they are not numerous.

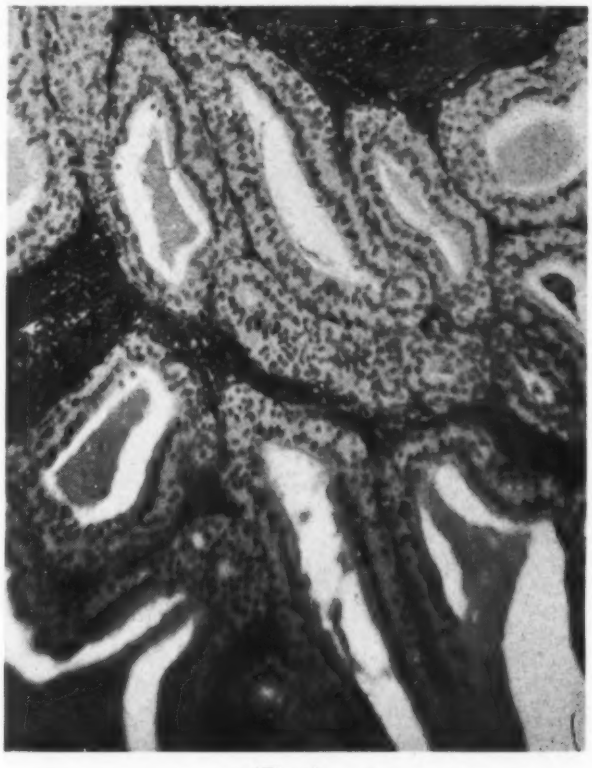


Fig. 6.

Warthin felt that these tumors were related to the branchial cleft cysts which are produced by heterotopia of pharyngeal mucosa. These, he suggested, were representative of heterotopia and dystopia of the eustachian tube mucosa to the parotid region. The term papillary cystadenoma lymphomatosum is used to describe this rare teratoid tumor.

CASE 7.—Tularemia.

"Enlarged submaxillary node." No other history available.

The lymph node was enlarged as the result of a hyperplastic lymphadenitis. In a few areas there were granulomatous masses of varying size, as shown in Figs. 7*A* and 7*B*. The granulation tissue was composed of young fibroblasts and many large mononuclear cells. There was evidence of only slight angioblastic proliferation. The central portions of the granulomatous masses showed focal necrosis and suppuration. There was a marked accumulation of epithelioid cells bordering the focal area. No epithelioid giant cells were present. These changes present the picture of tularemia which is an unusual finding in the lymph nodes of this area.

CASE 8.—Nevus and neurofibroma.

A white man, aged 35 years, came to the clinic because of a dark purplish-brown, verrucous lesion present on the lower lip. He stated the lesion had been present since birth and had increased slowly in size. Recently, he had the impression it had been enlarging. The history and physical examination were essentially negative except for the presence of the lesion on the lip. This lesion began at the angle of the mouth on the right side and involved about two-thirds of the lower lip. It involved the full thickness of the lip and



Fig. 7*A*.



Fig. 7*B*.

extended down to the mucobuccal fold and up on the gingiva to the free margin. On the skin surface, it involved about one-half the depth of the chin. The lesion was of a soft consistency and a deep purplish-brown color. On the gingival mucosa below the central incisors were two dark brown to black spots, 2 mm. in diameter.

Biopsy of the lip and gingival tissue revealed the character of the neoplastic process. Beneath a hyperplastic mucosa there were individual and small groups of large polyhedral cells with a light, slightly blue-staining cytoplasm. The nuclei were light staining because of the finely divided chromatin. In a few areas, these cells had an associated pigment characteristic of melanin. They appeared as typical nevus cells. These nevus cells were in a delicate connective tissue stroma. Deeper in the tissue these nevus cells blended into a tissue composed of delicate hyperchromatic spindle cells which appeared to be of nerve origin. They produced areas characteristic of the neurofibroma. Fig. 8 shows both types of cells.

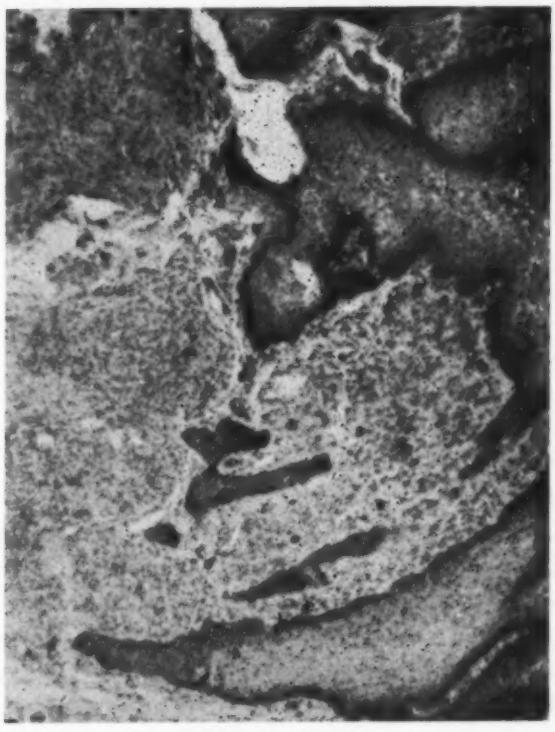


Fig. 8.

This case is of interest because of the frequent association between the nevus and the neurofibroma. It is also of interest because of involvement of the gingival mucosa, which is a rare finding.

The entire lesion was removed surgically. Recovery was uneventful and in four years the patient had shown no evidence of recurrence.

Because of the frequency with which cases are misdiagnosed from clinical findings alone, all cases should have a tissue examination. With information obtained from tissue sections, a more rational treatment can be outlined. From these few cases it can be seen that every case, regardless of its simplicity, may give us additional clinical and pathologic criteria upon which to base a diagnosis.

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DENTIGEROUS CYSTS

JAMES R. HAYWARD, D.D.S.

ARISING from the anomalous development of odontogenic tissue, the dentigerous cyst, although benign in itself, means potential destruction for contiguous structures. Incidence appears to be greatest in the first and second decades of life, but, commonly, insidious and asymptomatic growth masks detection until later. Dentigerous cysts may be found anywhere in the alveolar process, and their expansile destruction leads to marked bone deformities, possible pathologic fracture, and significant disturbances in dentition. Infection may complicate the process at any time.

By definition, a dentigerous cyst is an epithelium-lined sac which encloses the crown of an unerupted tooth or odontoma and is filled with fluid or semisolid matter. The anomaly appears to arise from degenerative and proliferative changes in the reduced enamel epithelium after the apposition of enamel. This characteristic has been emphasized by Robinson[4] in differentiating dentigerous cyst from follicular cyst, which commonly has been considered synonymous. Both develop from amelogenic epithelium: follicular cysts before, and dentigerous cysts after, enamel apposition. A true follicular cyst, therefore, would not contain enamel. Robinson[4] clarifies the distinction by replacing "follicular" with the term "primordial" in classification. This also eliminates the misleading association with the true dental follicle of odontogenesis.

Various theories have been advanced to explain the stimulus for dentigerous cyst formation. Inflammatory and chemical factors are apparently involved in many cases, but no explanation is complete for the entire field of dentigerous cysts. Borderline association with neoplastic adamantinoma stemming from the same anlage further complicates the etiology.

Early recognition and treatment of dentigerous cysts simplify management and prevent complications. Routine radiographs are often the first clue in diagnosis. Differential diagnosis is simple with the exception of unilocular adamantinoma. Thoma[5] points out notching of the bony wall and adjacent root absorption as radiographic signs of adamantinoma which may serve in differential diagnosis. The importance of pathologic diagnosis in handling these as well as our other surgical problems needs no additional emphasis.

Surgical management of dentigerous cysts may indicate complete enucleation or a Partsch operation. Enucleation is the treatment of choice when cysts are small and when the procedure will not sacrifice important teeth. Larger cysts may respond favorably to the Partsch operation, based on the fact that cystic epithelium may undergo metaplasia to become normal oral epithelium. In this manner the cyst is transformed into a recess of the oral cavity. When cystic pressure is removed, bone regeneration rapidly reduces the size of the recess. By utilization of cystic lining, epithelization of the defect is unnec-

essary. The operation employs unroofing of the cyst by sharp dissection, control of hemorrhage, irrigation, evacuation, and inspection of the cavity, and loose packing of the defect. The piece of excised cyst wall should be suitable for pathologic diagnosis. Wherever possible, the unroofed cyst should be thoroughly inspected. Mural nodules are signs of potential adamantinoma, emphasized by Cahn.[2] When signs of adamantinoma are present, enucleation should be complete and possibly followed by coagulation in an attempt to eradicate the neoplasm completely. Pyogenic granulation and papillomatous masses on the cyst wall also indicate enucleation as the treatment of choice. If the cyst wall appears to be of good tissue quality, indicating the Partsch operation, the procedure may be modified by suturing the incised margins of cyst membrane to the margins of normal oral mucosa (Fig. 10). Surgical management is influenced greatly by proximity of the cyst to the nasal cavity and maxillary sinus. The Partsch procedure precludes the possibility of oroantral and oronasal fistulas as complications.

In the course of postoperative treatment, various obtundent and antiseptic packs of decreasing size are used to keep the healing defect open and clean. Every three to five days the loose packs may be changed and the recess irrigated. A superficial closure of soft tissues is thus avoided. After initial healing the opening may be maintained by a modeling compound or acrylic plug which is made smaller as healing progresses from below. Regression of the cavity depends upon the size of the defect and bone regeneration of the individual. An average case may heal completely in six to ten weeks.

The following brief case reports illustrate clinical problems and variations in treatment:

CASE 1.—M. S., an 18-year-old white woman, presented an asymptomatic dentigerous cyst associated with an unerupted mandibular third molar detected upon radiographic examination during root canal therapy of a carious adjacent second molar. Patient's general health was good and past history and review of systems were essentially negative. All oral tissues were normal and only minimal hard swelling could be palpated in the retromolar and angle region. Radiographically the cyst appeared to be 2 by 3 by 3 cm., about the crown of the unerupted third molar situated near the inferior border of the mandible (Fig. 1). Under local anesthesia the cyst was unroofed and its semisolid brown paste contents evacuated. Inspection revealed an exceedingly thin cyst membrane of poor tissue quality and not adherent to adjacent tissues. It was felt that removal of the third molar was indicated and such procedures necessitated damage to the cyst lining. For these reasons the cyst wall was completely enucleated and the third molar removed. The inferior alveolar canal was identified along the floor of the cavity and was undisturbed. Coaptation of soft tissues with sutures partially closed the opening, and the cavity was packed with odontalgicum-petrolatum gauze. Pathologic diagnosis confirmed preoperative diagnosis of dentigerous cyst. No prolonged anesthesia or paresthesia was encountered, and convalescence was entirely uneventful (Fig. 2).

CASE 2.—R. L., a 22-year-old white woman, was referred after radiographic discovery of an asymptomatic dentigerous cyst, 2 by 4 by 2 cm., arising about the crown of a horizontal unerupted mandibular third molar (Fig. 3). History was noncontributory, and general health was good. At operation under local anesthesia, exposure and unroofing of the cyst revealed a heavy lining membrane of good tissue quality. Roots of the adjacent second molar were involved and removal of cyst wall would have entailed devitalization. Following

the Partsch procedure, the third molar was carefully removed and the cyst wall margins sutured to the edges of normal oral mucous membrane. A loose pack of odontalgicum-petrolatum gauze was inserted. Pathologic diagnosis confirmed original impression of dentigerous cyst. The second molar remained vital. Postoperative course was uneventful, and follow-up x-ray five months later showed recalcification and trabeculation (Fig. 4).



Fig. 1.



Fig. 2.

Fig. 1.—Case 1. Preoperative radiographic appearance.
Fig. 2.—Case 1. Two months after extraction and cyst enucleation.



Fig. 3.



Fig. 4.

Fig. 3.—Case 2. Preoperative radiographic appearance.
Fig. 4.—Case 2. Five months after extraction and Partsch operation.

CASE 3.—J. T., a 14-year-old white boy, presented complaint of a painlessly increasing bulge of the left body of the mandible during a four-month period. Otherwise, the condition was asymptomatic, and history was medically insignificant. Oral tissues were normal except the expansile, noninflammatory deformity of the buccal cortical plate palpated from the cuspid to the mesial of the second molar (Fig. 5). Lateral jaw films revealed a large cyst involving a malposed unerupted second premolar and a retained deciduous second molar (Fig. 6). Under local anesthesia, a horizontal incision was made just below the alveolar crest to the buccal between cuspid and molar. Reflection of the overlying mucoperiosteum revealed an eggshell-thin bony cyst roof, which was removed and fluid evacuated. No difficulty was encountered in removal of the second premolar, but the roots of the submerged retained deciduous molar were ankylosed, and complete removal was accomplished with surgical drills. Cyst membrane was believed nonviable and completely enucleated. Root surfaces of first premolar and first molar were exposed during operation, but their apices apparently were not involved. The cavity was packed with petrolatum-iodoform gauze dusted with ethyl aminobenzoate. Pathologic diagnosis of dentigerous cyst was made. Adjacent teeth remained vital. A patent fistula to the root surface of the first molar produced hypersensitivity, and a protective coat of precipitated ammoniacal silver nitrate was applied. Slight paresthesia of the lower left lip persisted for six months following operation. Convalescence was otherwise uneventful and the defect completely repaired.

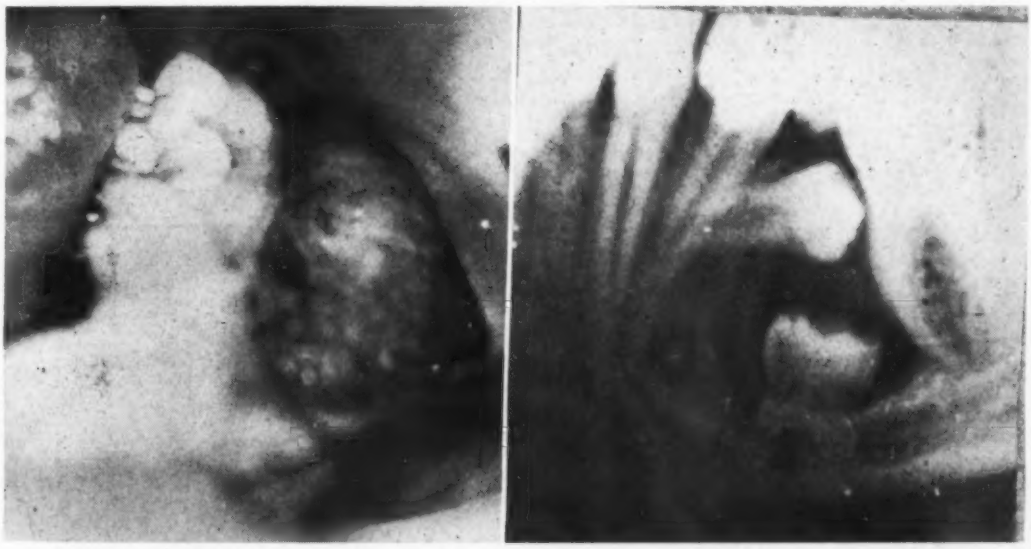


Fig. 5. Fig. 6.

Fig. 5.—Case 3. Obliteration of the vestibule by cyst tumescence.
Fig. 6.—Case 3. Preoperative radiographic appearance.

CASE 4.—F. D., an 8-year-old boy, was brought to the clinic by his local dentist after discovery of an asymptomatic dentigerous cyst involving the unerupted mandibular left first molar. Except for the usual childhood diseases past history was negative and irrelevant. Mandibular deciduous second molars had been removed two months previously after carious exposure. Facial asymmetry was minimal with palpable tumescence of the buccal cortical plate from mandibular left cuspid to a point 2 cm. anterior to the angle. All oral tissues were normal, and, posterior to deciduous first molars, the mandible was edentulous. The bulging deformity had completely obliterated the vestibule in this region and slight crepitus was produced by firm palpation. X-ray examination demonstrated the cystic

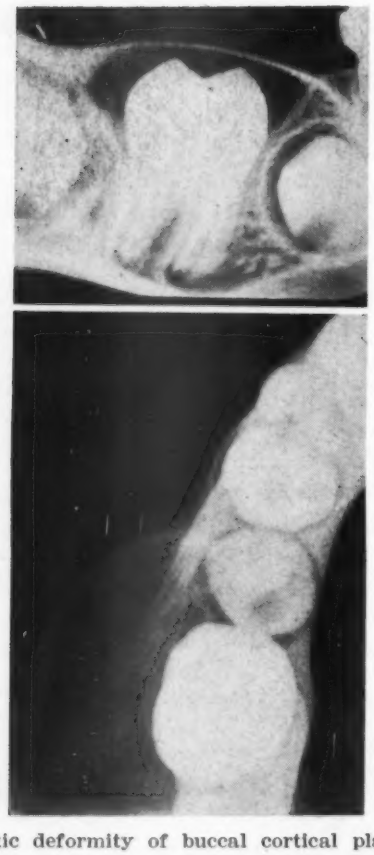


Fig. 7.—Case 4. Cystic deformity of buccal cortical plate radiographically.



Fig. 8.—Dentigerous cyst involving second premolar, managed as in Case 4, showing stages in eruption which followed.

envelopment of the unerupted first molar (Fig. 7). At operation under local anesthesia a section of overlying alveolar ridge was removed exposing the cystic cavity. The defect extended inferiorly on the buccal to the inferior border of the mandible. The cystic lining was heavy and of good quality. No fluid was identified. The unerupted first molar was undisturbed, the cavity was exposed widely buccally, and a pack of odontalgicum-petrolatum gauze inserted. The cyst wall was described microscopically to contain areas



Fig. 9.—Dentigerous cyst associated with a supernumerary tooth. Treated by Partsch procedure.

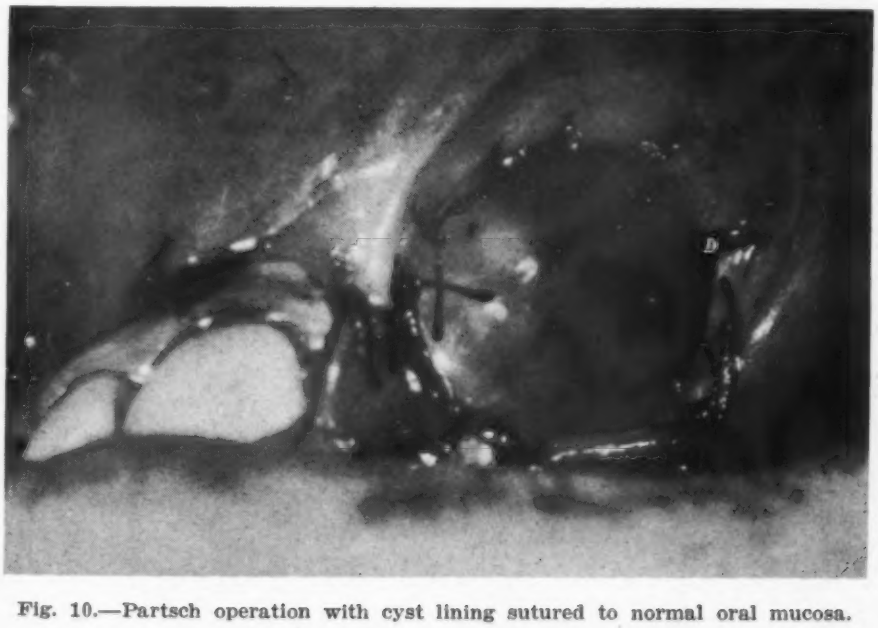


Fig. 10.—Partsch operation with cyst lining sutured to normal oral mucosa.

of myxomatous connective tissue invaded by epithelium in a manner suggesting adamantinoma. Postoperative course was uneventful with filling of the defect and eruption of the first molar in position. The question of adamantinomatous change in the cyst wall necessitates an intensive and prolonged postoperative observation to detect signs of recurrence.

SUMMARY

Following a brief review of dentigerous cyst etiology and description, four cases have been presented to illustrate clinical problems and variation in surgical management. It is important to recognize the indications and contraindications for enucleation and the Partsch operation.

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THE ODONTOMAS

Two Case Reports

Jerry K. Jacobson, D.D.S.

MUCH interest has been shown in the diagnosis and treatment of the odontomas. The varied and bizarre forms which this tumor takes to go alter the treatment considerably in the many instances of its occurrence. Whether radical or conservative procedures are indicated depends upon the form which the particular case presents.

Odontomas are produced by abnormal development of the tooth bud or from supernumerary buds formed by the dental lamina. The process of development of the normal tooth buds has been very adequately described in most textbooks of dental histology.

The classification of Bass[1] is very simple and adequate in its description of odontomas and embraces all of the various forms of odontomas that are found, taking into consideration the tissues from which the tumor arises, as:

1. Epithelial odontomas—which are derived from the enamel organ, or epithelial portion of the tooth germ, during a period of abnormal development or from epithelial rests. This group is composed of the adamantoblastomas, the dentigerous cysts, and the cystic adamantoblastomas, and can be construed to encompass the enameloma, which Bass himself does not consider to be a true odontoma.

2. Composite odontomas—which are derived from the enamel organ and the dental papilla, or connective tissue portion of the tooth germ, during a period of abnormal development. These odontomas are composed then of both epithelial and connective tissue elements with or without calcification. These are the complex composite odontomas and the compound composite odontomas.

3. Connective tissue odontomas—which are composed entirely of connective tissue elements as the soft odontomas, dentinomas, and cementomas.

Whether or not the odontoma is soft or hard depends upon the period of development of the tooth germ at which the aberration occurs. The earlier in the developmental period, the less deferentiated will be the cellular details and the lesser will be the degree of calcification. If abnormal development occurs during the first stages of differentiation, the adamantoblastomas may be formed. These tumors which develop earlier in stages of differentiation have much greater tendencies toward malignant degeneration.

The tumors which develop later during the process of tooth germ differentiation and calcification are the hard odontomas which are more commonly described. They are of local significance and have no malignant tendencies. These odontomas may be formed by a single particle of concrescence or several hundred particles in one mass. The size may vary from a minute denticle of a millimeter or less in diameter to a size several centimeters in diameter. Crecca, et al.,[5] describe an odontoma 35 by 25 by 20 mm. Larger ones have been reported. They are characterized by masses of calcific material of the nature of tooth structure and are usually surrounded by a fibrous capsule.

Aberration may occur during several periods in development of a single tooth germ and thus form types of odontomas with both hard and soft characteristics. The mixed adamanto-odontoma described by Kemper and Root[8] is an illustration of such a tumor.

Ewing[6] states that the typical odontoma occurs almost invariably in the mandible. They are also found very frequently in the maxilla. It is difficult to say whether the tumors occur more frequently in the anterior or posterior areas of the jaws, but it appears that they are slightly more common in the posterior areas. The apparent increase in frequency of occurrence in the posterior regions may well be due to the crowded, confined area in which the tooth germs are forced to develop.

The clinical appearance of the odontoma depends upon its type, situation, and whether or not infection is present. The adamantoblastoma is usually found in the posterior regions of the mandible or maxilla and is usually asymptomatic until it reaches rather large proportions or becomes infected. In the event that infection occurs, the usual signs and symptoms of inflammation are present, and a discharging fistulous tract may be formed. Less commonly, there may be painless swelling, and on rare occasions there are complaints of vague pains over the area of distribution of the components of the trigeminal nerve on the affected side. The adamantoblastoma is in many cases only of local significance and, if small enough, complete enucleation is feasible and the treatment of choice. The confines of the resulting bone defect should be cauterized in order to insure removal of all cells which might potentially produce recurrence. The region should be observed periodically by radiographic means, and if recurrence is noted, the entire area should be resected. If malignant properties are present at the time of first discovery of the tumor, wide excision of the bone and tissue well outside the boundaries of the lesion should be carried out without any attempt at enucleation.

The hard odontomas are of local significance, very slow growing, and rarely develop after completion of formation of the second or permanent dentition. They are usually associated with missing or unerupted teeth. Odontomas arising in areas from which a tooth is missing can be considered to have developed from aberration of the tooth germ of the absent tooth. Odontomas which obstruct eruption of normal teeth can be considered to have arisen from aberration of supernumerary tooth germs, epithelial rests, or from the tooth germ of the unerupted tooth after completion of the development. Clinically, however, all of the normal teeth may be present. The alveolar process may be firm and apparently normal or it may be somewhat tumescent and the overlying soft tissues are apt to be of normal color, tone, and density. Large swellings due to cystic degeneration or infection have not been uncommon in the past, but with increased use of the radiograph the odontomas are being discovered earlier and the large swellings are less frequent. Again, there may be symptoms of dull pains of varying intensity over the affected side of the jaw or head and, if infection is present, there are the classical signs of inflammation.

Treatment of the hard odontoma consist of complete removal of the tumor by enucleation. If removal is incomplete, recurrence may be expected. Should

recurrence be noted, further enucleation should be carried out. There is no apparent necessity for radical resection in these cases because they do not have malignant tendencies. Upon being exposed, these tumors are usually found to have a fibrous capsule which separates them from the surrounding bone. This capsule must be removed in its entirety if recurrence is to be prevented.

The cases to be presented are composite odontomas of the complex type.

CASE 1.—C. A., a Negro man 37 years of age, entered complaining of dull aching pain in the left anterior region of the maxilla. The pain had been of two years' duration, and of increasing severity. The use of mild sedatives gave no relief. The patient stated that he had been in an automobile accident sixteen years previously, at which time a left maxillary incisor was knocked out. He was not certain as to whether the other incisor was driven up into the alveolar process or was also knocked out. The area had been asymptomatic until onset of the present illness.

Examination revealed a well-developed, well-nourished Negro man without obvious abnormality about his head, face, or neck, and no palpable regional lymph nodes. The maxillary left central and lateral incisors were absent. The remaining teeth appeared to be in good state of repair. The oral soft tissues were essentially negative, being of good color, tone, and density. No swellings or areas of tenderness were noted. A supernumerary tooth was evident in the maxillary right molar area distal to the third molar. Radiograms revealed the missing left maxillary central incisor to be embedded in the alveolar process just to the left of the midline with the long axis of the tooth directed parallel to the sagittal plane (Fig. 1). Superimposed and to the left of the crown of this tooth could be seen several calcified bodies appearing to be composed of tooth structure. These appeared to be supernumerary teeth or denticles. A supernumerary tooth was also seen in the maxillary right molar area (Fig. 2).

Treatment.—Under local anesthesia a mucoperiosteal flap was raised from the labial surface of the maxillary anterior alveolus, and the labial plate over the edentulous area was removed. The fibrous tissue capsule was opened when it was found impossible to remove the entire mass in toto. Many of the denticles were found to be embedded in individual sockets in the alveolar bone with their coronal portions connected by the fibrous tissue. All of the denticles together with the impacted central incisor were removed, as was the fibrous capsule. The edges of the bony defect were smoothed and the soft tissues reapproximated by use of black plastic silk sutures. A gauze sedative dressing was inserted into the operative area for control of postoperative pain.

The patient's postoperative course was uneventful, and the area filled in quite satisfactorily after a period of three weeks. The preoperative symptoms were completely relieved. Postoperative radiograms revealed no evidence of odontoma (Fig. 3). Twenty-one denticles in all were removed from the operative site (Fig. 4). As has been stated previously, odontomas composed of more numerous denticles have been found.

CASE 2.—J. V. F., a white man 36 years of age, had a complaint of dull pain in the right side of his mandible of two months' duration. The pain had increased in intensity and become continuous. The usual sedatives had been of little aid in alleviation of his symptoms.

Examination revealed a well-developed, well-nourished white man in apparent good health, although he appeared older than the stated age. There were no questionable lesions about his head, face, or neck, and no enlarged palpable lymph nodes. Oral examination revealed the maxilla to be edentulous, with signs of recent extraction of teeth on the left side. The mandible was partially edentulous. The right mandibular canine was rotated out of normal position. The soft tissues of the mouth were essentially negative. At the labiogingival fornix in the mandibular right canine region a prominence of the labial plate of the alveolar process could be palpated. It was hard, smooth, nontender, and the overlying tissues were negative. Radiograms revealed a mass of calcified bodies about the radicular areas of



Fig. 1.—Radiographic appearance of affected region showing impacted maxillary left central incisor and odontoma of Case 1.

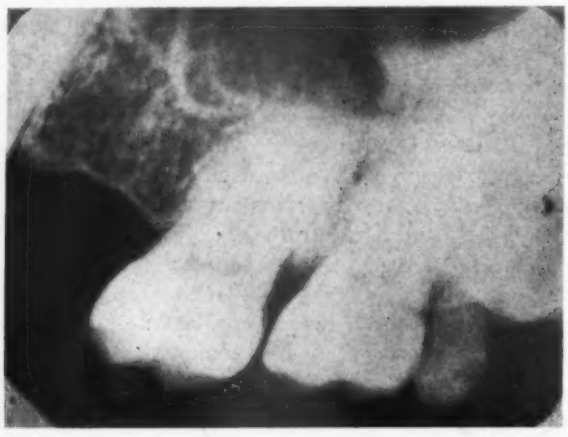


Fig. 2.—Maxillary right molar area with supernumerary tooth distal to third molar in Case 1.



Fig. 3.—Postoperative radiograph of affected area of Case 1 showing complete absence of odontoma and impacted left central incisor.

the mandibular right central and lateral incisors and canine tooth (Fig. 5). Radiographically the mass appeared to be composed of tooth structure.

Treatment.—Under local anesthesia the remaining teeth in the right mandible were removed. A mucoperiosteal flap was raised in the canine region, and the labial plate was removed to expose the hard tumor mass. The denticles were removed, complete with the

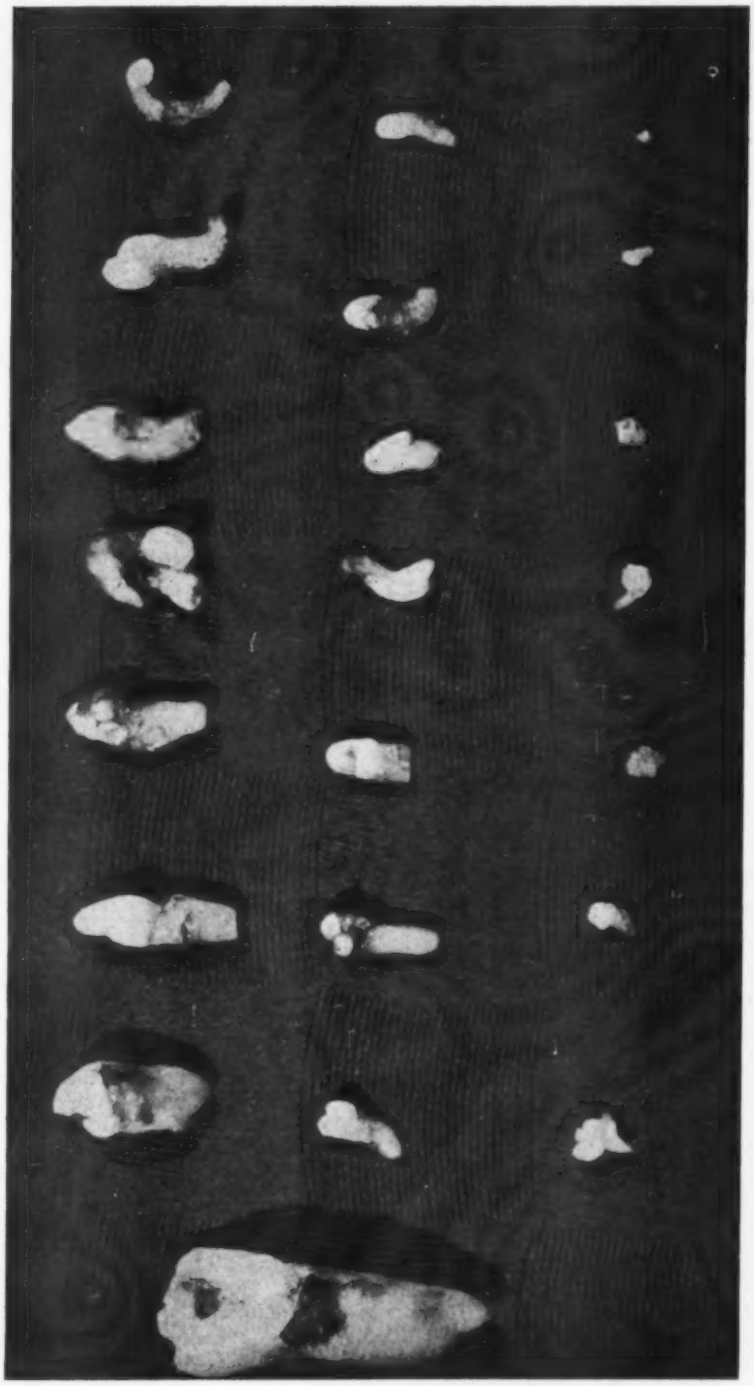


Fig. 4.—Incisor tooth and 21 denticles removed in Case 1.

fibrous capsule. The bone was smooth, and sulfanilamide powder was insufflated into the area. The soft tissues were reapproximated by use of black plastic silk interrupted sutures. A gauze sedative dressing was inserted into the operative area for control of postoperative pain.

The patient's postoperative course was uneventful, with complete healing of the area. The preoperative symptoms were completely alleviated.



Fig. 5.—Radiographic appearance of odontoma in Case 2.

SUMMARY

Odontomas are formed by aberration in the development of the normal or supernumerary tooth germs formed by the dental lamina and papilla.

The odontomas can be classified according to the tissue from which they originate.

The tendency toward malignancy depends upon the stage of development of the tooth germ at which aberration occurs.

Symptoms may be entirely lacking or profound. Use of the radiograph has led to earlier discovery of odontomas.

Treatment of the odontoma is complete enucleation.

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The Diagnosis, Surgical Management, and Histopathology of Selected Interesting Clinical Cases

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INTRODUCTION

Interdependence

PROBABLY the most difficult educational problem confronting deans of dental schools, and, to at least an equal extent, deans of medical schools, is the question of how to bring about a unification in the student's mind of subjects he has been studying in various isolated departments. If such unification is not achieved, much of the time spent taking science courses is almost as purposeless as the human sacrifice made by primitive people to their otherwise implacable gods. The only difference is that we sacrifice only a few years of our young people's lives and that these sacrifices are now made to the god Academic Custom and his consort Licensure.

This condition is well exemplified in the field of surgical instruction. In spite of a natural liaison which exists between surgery and pathology, x-ray and other divisions of the dental curriculum, this subject is often taught as if it were a glorious and independent art and all too frequently the impression is given that the purpose of the associated science is but to serve for the greater glorification of surgery. Since few surgeons are distinguished by their knowledge of or interest in physiology and other basic sciences, the problem is being accentuated by placing the instruction of oral surgery, exodontia, and anesthesia in the hands of men who are specialists in these fields. The growing tendency toward using hospitals for surgical instruction is increasing the gap between surgery and the preclinical science departments.

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Obviously the problem of training dentists who can carry into their practice the maximal amount of usable scientific background is not susceptible to a simple solution. Perhaps the most that can be done is to suggest a principle which could serve to guide deans and departmental heads alike: this is, that no department is discharging its full responsibility unless it is also aiding instruction in other divisions of the school. In surgery this end can be approached by avoiding overdepartmentalization, and by encouraging interdepartmental cooperation in research or on case studies of the sort reported in this issue of the JOURNAL. Occasions to use anatomic sections and microscope demonstrations should be found by clinical teachers, but, most important of all, it is necessary that the instruction be placed in the hands of men who have the broadest possible scientific and educational interests.

B. G. B.

I. TRAUMATIC CYST OF THE MANDIBLE

JOSEPH P. LAZANSKY, M.D., D.D.S., D.M.D., ARTHUR H. WUEHRMANN, D.M.D., AND IRVING GLICKMAN, B.S., D.M.D.

IN FEBRUARY, 1945, a 16-year-old girl of Greek extraction presented herself for routine dental treatment at Tufts College Dental School. Full-mouth x-rays were taken and a large radiolucent area was noticed in the region of the lower incisors. Occlusal films disclosed a cystlike radiolucent area that extended from cuspid to cuspid. Questioning of the patient elicited the usual history of childhood diseases, chickenpox, and scarlet fever. The mother of the patient was able to recall that about four years previously, April, 1941, the patient fell on her face. As a result of this accident the upper right central incisor was fractured slightly but did not give rise to any serious complications, and pain was present only when cold or heat was applied to the tooth. This discomfort lasted for one week. No evidence of pain or swelling in other parts of the jaws was noticed.

On clinical examination the patient's teeth were found to be relatively free from caries. There were no external signs of injury of the upper or lower front teeth, except for an insignificant fracture of the distal edge of the upper right central incisor. On transillumination, all anterior teeth (upper and lower) seemed to be normal. The vitality of all the upper and lower front teeth was tested with the Burton Vitalometer and gave values at normal levels. All these teeth were firm, had normal alignment and color, and were noncarious.

X-ray examination revealed a large, sharply defined radiolucent area beneath the roots of all the lower six anterior teeth, without abnormal surrounding bone condensation. The apices of all the teeth showed an intact lamina dura (Fig. 1). These facts as well as the vitality of all the lower six teeth apparently excluded the possibility of a radicular cyst. The absence of bone condensation around the area and the normal number of teeth supported the belief that our case was not a follicular cyst, as did the existence of a normal bone configuration (no expansion of the cortical bone). The history of trauma supported the tentative preoperative diagnosis of a traumatic cyst, as against a localized osteitis fibrosa cystica. The sharp outline of the radiolucency strengthened this diagnosis.

The laboratory findings (blood calcium, phosphorus, and phosphatase) were essentially negative.

An operation was performed on April 30, 1945. Under bilateral mandibular block, a flap was raised from the lower right first premolar to the lower left first premolar. There was no resorption of the buccal plate and the cavity had to be opened with mallet, chisel, and rongeur. The cavity itself was partially filled with a dark, almost black fluid. The roots of all the lower teeth were well covered with bone (Fig. 2). On attempting to curette the cavity, a cob-

weblike structure, probably fibrin, could be detached from the walls, but it was impossible to remove it in its entirety. The portion of the cavity most removed from the roots was curetted and some of the lining and bony material retained for pathologic examination.

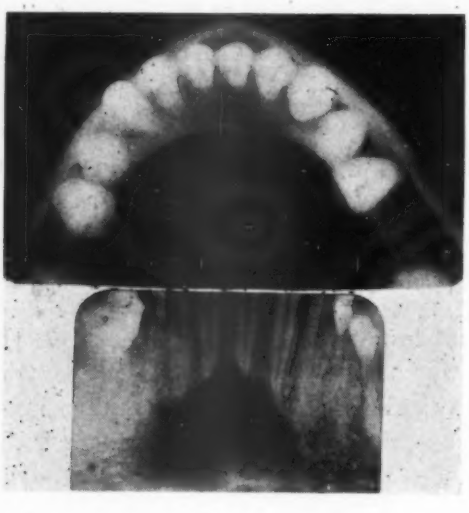


Fig. 1.

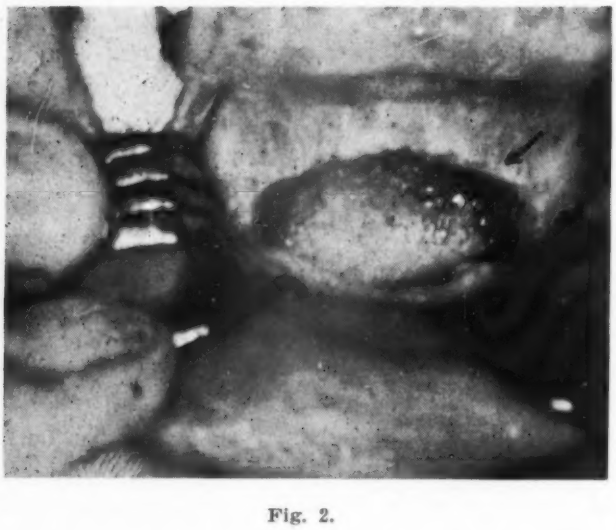


Fig. 2.

The cavity was cauterized with phenol, followed by alcohol, and closed with seven interrupted sutures. An iodoform gauze dressing was inserted between two sutures. Finally, 5 c.c. of penicillin (500 Oxford units per cubic centimeter) were injected into the cavity.

The patient was seen on May 2, 1945. At this time there was some pain and a slight swelling of the lower lip which was probably of traumatic origin. The dressing was removed and the penicillin therapy repeated. Subsequently the patient was re-examined at the dates listed below and the following observations were made:

May 7, 1945: Sutures were removed. Lower lip was normal and all swelling had disappeared.

May 18, 1945: Wound had closed completely. Control x-rays were taken.

Vitality test—$\overline{3\ 2\ 1}\,|\,1\ 2\ 3$*

0 0 0 0 0 7

July 18, 1945: Vitality test—$\overline{3\ 2\ 1}\,|\,1\ 2\ 3$*

5 0 0 5 0 5

Sept. 29, 1945: Vitality test—$\overline{3\ 2\ 1}\,|\,1\ 2\ 3$*

4 6 0 6 0 7

Jan. 8, 1946: Vitality test showed all teeth vital.

Control x-rays showed a marked reduction in radiolucency and new trabeculae forming in the bone cavity (Fig. 3).

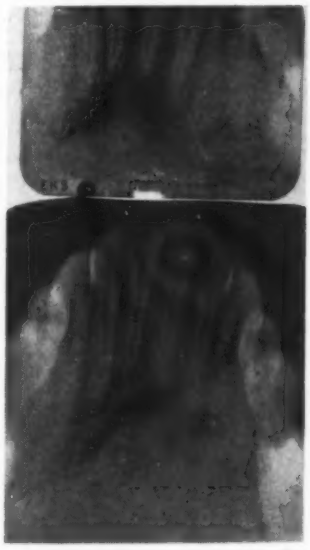


Fig. 3.

Pathologic examination (Department of Oral Pathology, Tufts College Dental School): Specimen consists of a section of dense cortical bone. The osteocyte lacunae are intact and contain osteocytes which are normal in appearance. In some areas the vessel channels are lined by a thin layer of bone which is demarcated from the remaining bone substance by a deeply staining linear lamellar marking. There are no osteoclasts. There is no evidence of malignancy.

DISCUSSION OF DIAGNOSIS

Traumatic bone cysts (hemorrhagic, extravasation cysts) are probably a result of trauma, and all reported cases have occurred in young people. The traumatic cysts of the jaws were first mentioned and described in this country

*Numbers shown above are symbols for the tested teeth. Numbers shown below represent the values found for each tooth with the Vitalometer.

by C. J. Lukas.[1] Later, Schneider[2] and Blum[3] described the pathology of this condition. It also occurs in the long bones, where it was studied by Haberer[4] and Pommer.[5] Subsequently, cases were reported by Thoma,[6, 7] Seldin,[8] Ivy and Curtis,[9] Smith and Barrows,[10] and Fink.[11] Most authorities are in agreement on the pathogenesis of these cysts which occur in young people due to injury "insufficient to cause fracture or escape of blood into the surrounding soft tissues."[12] The hematoma so formed exerts a pressure against the walls of vessels supplying the bone in the neighborhood of the affected area, preventing or inhibiting the resorption of blood. The products of the decomposing blood irritate the surrounding bone and cause its destruction.

Traumatic cysts are encapsulated by a thin fibrous membrane simulating endosteum. However, the wall may be of a thickened consistency, having an increased amount of cellular edematous connective tissue.

In general, traumatic cysts can be differentiated from odontogenic cysts by (1) the absence of a cyst membrane which can be removed in toto, (2) the lack of an epithelial lining (unless epithelium has grown into the cyst through a fistular opening), (3) the absence of bone expansion and the rare involvement of the cortical layer, and (4) the fact that in uncomplicated cases the teeth are not involved. The differential diagnosis of traumatic cysts from localized osteitis fibrosa cystica is determined by the fact that in traumatic cysts (1) the surrounding bone does not show indications of marked bone destruction or formation, and (2) the destroyed bone is not replaced by a pathologically altered cancellous bone.

COMMENT ON TREATMENT

Special attention was given in this case to the preservation of the vitality of the teeth which were in close proximity to the bone cavity.

It was felt that "wholesale devitalization of all teeth apparently roentgenologically involved in the pathological process is extremely radical, unnecessary and out dated."[13]

The operative procedure was planned with the thought of saving the teeth and of maintaining their vitality. After the operation all of the lower anterior teeth with the exception of the lower left cuspid gave no response to the pulp tester. Two months later, two additional teeth responded to the electric stimulation, and about four months after the operation, all except one ($\overline{1}|$) gave a positive reaction. In the final test on Jan. 8, 1946, all the teeth reacted normally.

Kaletsky[14] has shown that the vital response of the teeth may be missing if the teeth are tested shortly after the operation and that the vitality will show a gradual return to normal. If it does not return, root canal therapy can be attemped at a later date.

It should be emphasized that in postoperative cases lack of response to the pulp tester is not identical with pulpal death. During the operation we may injure or sever the nerve supply of the pulps without cutting off the blood supply; the vessels and nerves may reach the apical foramen by alternate pathways. The blood vessels have collaterals which the nerves lack, and, in the latter, regeneration is notoriously slow.

Regeneration of the injured nerves takes time and any undue hurry to begin a root canal treatment may be a disservice to the patient. If, on transillumination and observation, the color of the teeth compares favorably with the unaffected neighbors, there is every reason to expect a favorable prognosis after operation.

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II. LOCALIZED OSTEODYSTROPHY OF THE MANDIBLE AND MAXILLA

Case Analysis

Arthur H. Wuehrmann, D.M.D., and Irving Glickman, B.S., D.M.D.

THE diagnosis of osseous lesions of the mandible and maxilla which are unusual in radiographic appearance presents a challenge. In addition to their possible derivation from local pathologic changes, such lesions of the jaws may occur as manifestations of a generalized skeletal dystrophy or systemic disease. Because of the latter possibilities, the diagnosis of the less common osseous lesions of the maxilla and mandible require a general case history, physical examination, pertinent laboratory tests, and x-ray survey of other bones, in addition to a complete study of the jaws, and, whenever possible, microscopic examination of biopsy specimens. An analysis of the following case is presented here, first, because of the unusual nature of the radiographic findings in the mandible and maxilla, and, second, because it demonstrates the procedures necessary in attempting a diagnosis of such findings.

The patient, an edentulous 34-year-old woman, presented at the Tufts College Dental School for the purpose of obtaining full upper and lower dentures. Her teeth had been removed a short time previous to her admission to the dental school.

Clinical oral examination revealed no unusual findings.

Intraoral radiographic survey (Fig. 1) revealed the presence of one retained root in the left maxillary tuberosity, multiple areas of condensation throughout the maxilla and mandible, and one area of radiolucency in the left mandible in the first molar region. The radiopaque areas numbered approximately nine, and varied in size from 3 to 16 mm. Each area was irregular in shape, did not resemble any dental structure, had the density of fairly compact bone or of dentine or cementum, and was demarcated from the remaining alveolar bone by an irregular radiolucent peripheral zone. The single radiolucent area was spherical in shape, with a diameter of approximately 12 mm. It had an irregular contour with irregularly shaped radiopaque projections of varying density extending for a slight distance from the periphery into the radiolucent area, giving the margin an escalloped appearance.

Differential Radiographic Diagnosis.—Radiopaque areas: (1) Multiple odontomas. (2) Multiple osteomas. (3) Multiple areas of condensed bone following trauma or infection. (4) Paget's disease. (5) Multiple osteomas associated with hereditary deforming chondrodysplasia. (6) Syphilitic osteitis.

Radiolucent area: (1) Radicular or follicular cyst. (2) Traumatic cyst. (3) Adamantinoma. (4) Localized osteitis fibrosa.

General history disclosed that the patient had been receiving x-ray and radium therapy treatment for a Grade II epidermoid carcinoma of the cervix, previous to having submitted to a hysterectomy operation which was followed by an uneventful recovery. There was no history of any other operation, or of lung, kidney, heart, or diabetic disease.

The patient's father died at the age of 64 years, cause of death unknown. The mother is in good health at the age of 70 years. The patient has two brothers and sisters in apparent good health and had a sister who died at the age of 28 years, cause of death not definitely known to patient.

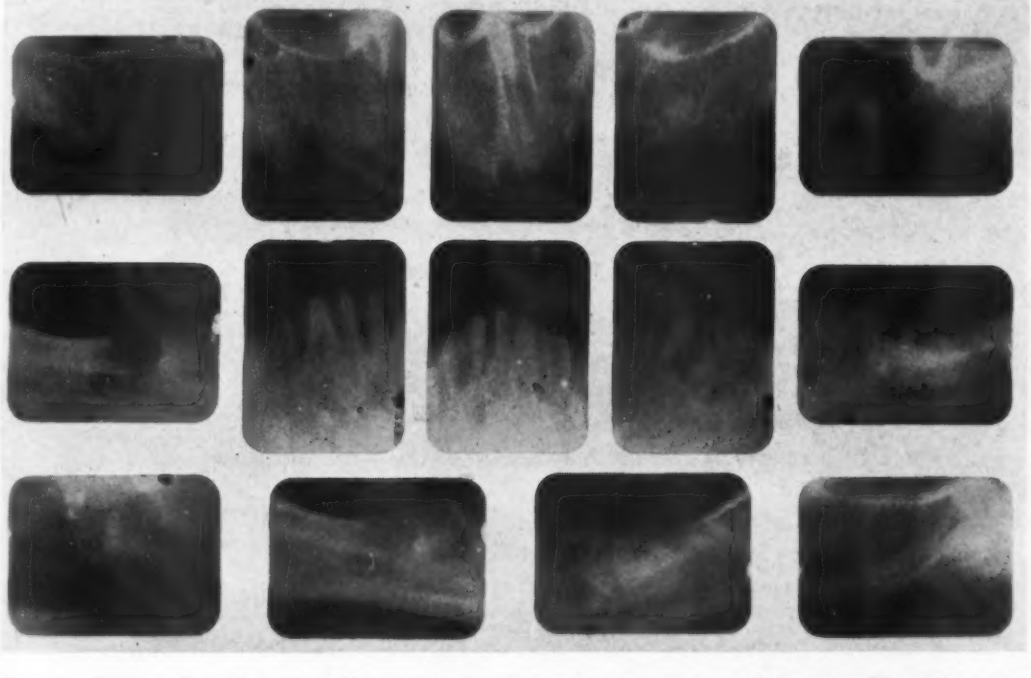


Fig. 1.—Intraoral series of radiographs showing root remnant, radiolucent and radiopaque zones.

Radiographic findings in the tibia, fibula, radius, ulna, and skull were negative.

Laboratory Tests.—Urine: cloudy, light amber, acid, specific gravity 1.018, no albumin, etc. Blood: Rh positive, blood pressure 120/60, hemoglobin 12 (15 = 100 per cent), red blood cells 4,500,000; differential: polys 69 per cent, lymphocytes 19 per cent, monocytes 10 per cent, basophiles 2 per cent, eosinophiles 2 per cent. Hinton negative. Calcium 8.5 mg. per cent. Phosphorus 3.1 mg. per cent. Alkaline phosphatase 2.5 units.

The radiolucent lesion* (*A*) and the largest radiopaque lesion (*B*) were removed surgically. The physical condition of the patient did not warrant additional surgery necessary for the removal of the lesions in the maxilla. Biopsy specimens were submitted for microscopic study.

*Operation performed by Richard H. Norton, D.M.D., D.D.S., Professor of Oral Surgery, Tufts College Dental School.

Microscopic Findings.—

A. Radiolucent lesion (Fig. 2): Section consists of a mass of cancellous bone one aspect of which is covered by a densely collagenous strip of connective tissue (Fig. 3). The remaining peripheral surface of the section is irregularly outlined and formed by alternate areas of bone and bone marrow. The bony trabeculae enclose marrow spaces containing a loose fibrous reticulum of stellate-shaped cells and anastamosing collagen fibers. Elongated spherical cells in close apposition to the inner bone surfaces comprise the endosteal lining for these marrow spaces.

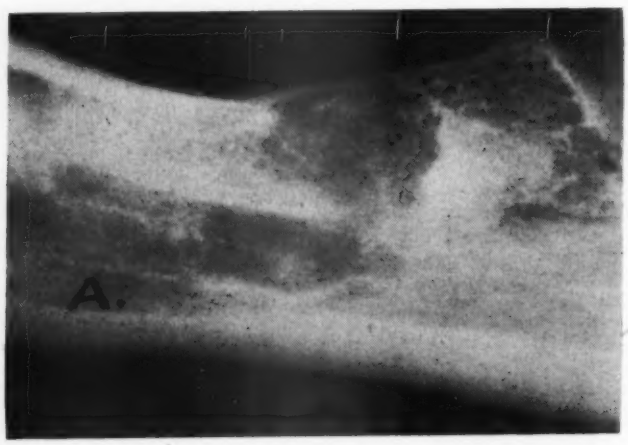


Fig. 2.—Radiograph showing radiolucent zone in left mandibular region.

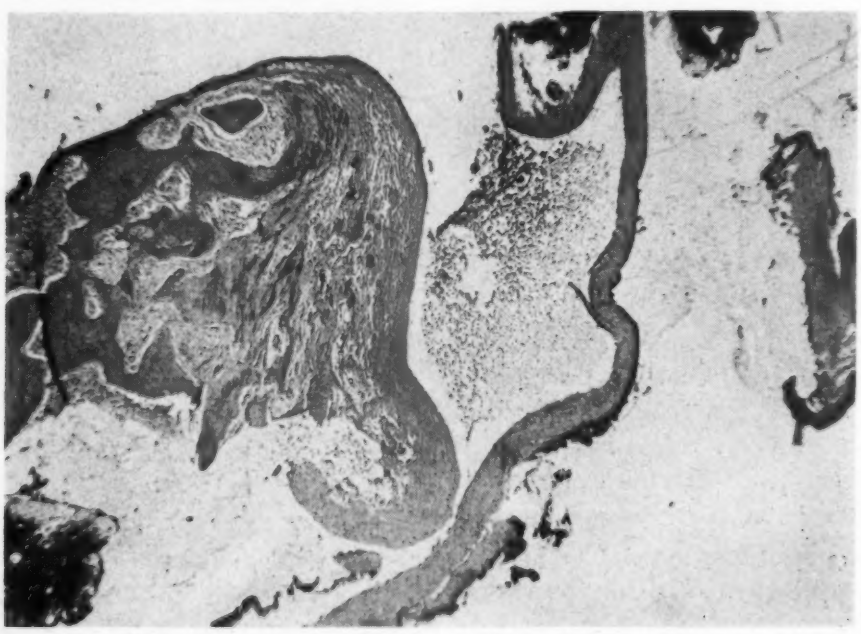


Fig. 3.—Low-power section showing relation of external connective tissue membrane to underlying bone. (Hematoxylin and eosin, ×50.)

The bone morphology varies in different areas of the section. In one area the trabeculae are relatively thin and are comprised of a comparatively pale-staining bone matrix with irregularly arranged large osteocyte lacunae which contain prominent ovoid osteocytes (Fig. 4). Polyhedral osteoblasts are seen arranged in linear manner along the borders of these trabeculae. Multinuclear

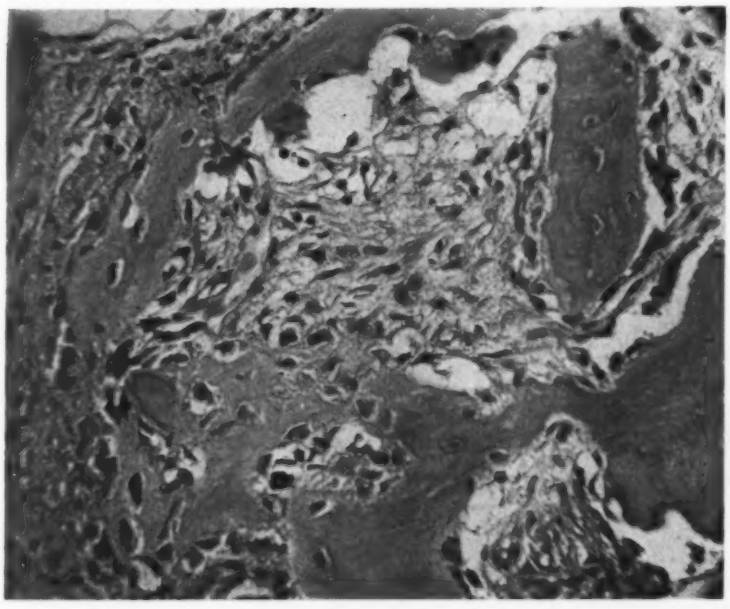


Fig. 4.—Newly formed bone trabeculae showing osteoblasts partially embedded in bone matrix and partially covered by connective tissue. Osteoclasts also seen. (Hematoxylin and eosin, ×425.)

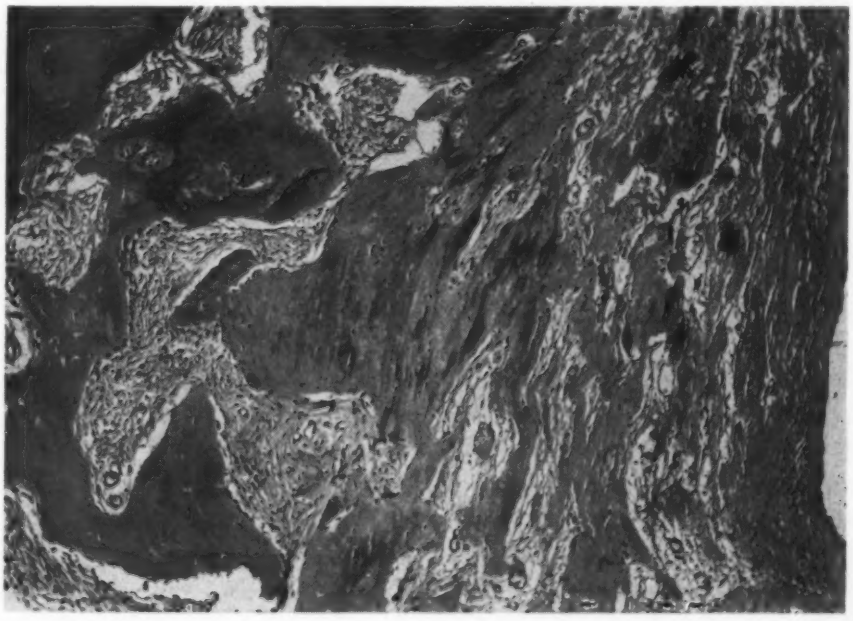


Fig. 5.—Detail of Fig. 3, showing change in bone matrix as outer connective tissue is approached. (Hematoxylin and eosin, ×215.)

osteoclasts are an occasional finding, partially enclosed within slight indentations in the bone surface. The bone trabeculae throughout the remainder of the section are wide in comparison with those previously described and present a marked variation in appearance in different regions. In the portion of the specimen farthest removed from the surface connective tissue, the bone matrix presents a suggestion of faintly demarcated lamellation and contains ovoid lacunae with deeply staining osteocytes. As the distance to the outer connective tissue capsule progressively diminishes, there is a gradual alteration in the appearance of the bone (Fig. 5). The matrix appears granular, fragmented, and fibrillar. The outlines of the osteocyte lacunae are distorted and in many instances osteocytes are absent or shrunken and pyknotic. Immediately beneath the connective tissue the bone appears as an amorphous, pale-staining mass, with markedly irregularly fragmented margins, and distorted, empty spaces suggestive of altered lacunae (Fig. 6). There is an imperceptible blending between the altered bone matrix and the fibers of the overlying connective tissue capsule.

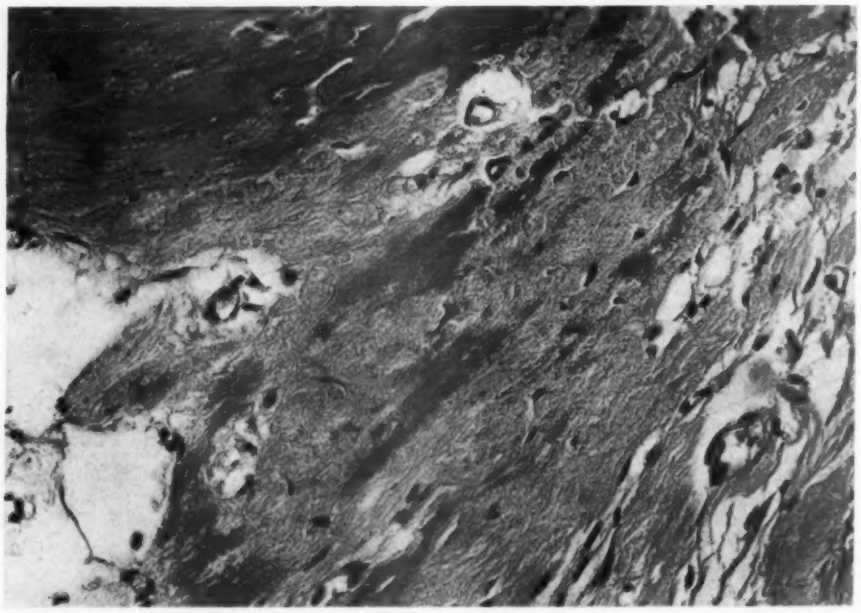


Fig. 6.—Detail of Fig. 5, showing fragmentation of bone, destruction of osteocyte lacunae, and blending of connective tissue and bone. (Hematoxylin and eosin, ×420.)

The external connective tissue capsule is comprised of closely arranged, densely packed, parallel bundles of collagen, interspersed along which are many deeply staining, flattened nuclei of connective tissue cells.

Several features of this lesion are noteworthy. The mass is apparently enclosed within a cystlike connective tissue membrane. There is a loss of bone substance and its replacement by connective tissue is seen. The bone presents a granular fragmentation of the matrix with destruction of the osteocyte lacunae and release of the osteocytes. In those areas in which the bone has undergone most marked changes, the altered bone and adjacent connective tissue blend

imperceptibly so that no line of demarcation exists between them. This replacement of bone by connective tissue accounts for the radiolucent appearance of the lesion radiographically.

In contrast with these findings, bone formation with osteoblastic activity and newly formed trabeculae are seen in another region of the lesion. The osteoblasts appear to develop from the connective tissue cells within the marrow spaces. The newly formed bone trabeculae replace the connective tissue.

The basic over-all nature of this lesion is one of a localized metaplastic equilibrium between bone and connective tissue. Although there is evidence in both directions of this equilibrium, the change from bone to connective tissue is the predominant one.

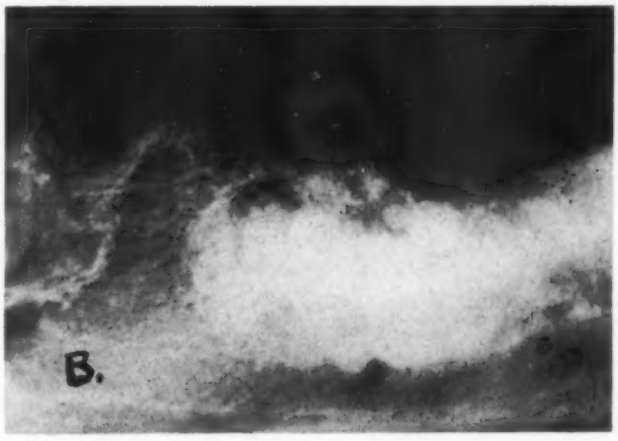


Fig. 7.—Radiograph showing radiopaque area in right mandibular region.

B. Radiopaque lesion (Fig. 7): Section consists of an irregularly shaped mass of closely arranged bone trabeculae with irregular peripheral outline formed by alternating sections of bone and bone marrow (Fig. 8). Enclosed within the trabeculae are many marrow spaces containing a loose meshwork of connective tissue cells and fibers. For the most part the bone matrix throughout the section presents irregular curvilinear lamellar markings and heterogeneously arranged elliptical osteocyte lacunae with enclosed osteocytes. The margin of the bone bordering the marrow space varies in different areas. In some regions a thin, single layer of flattened elliptical cells in apposition with a smooth bone margin comprises the endosteum of the marrow space. In other areas the cells lining the marrow space are polyhedral and deeply staining and are separated from the underlying bone matrix by a pale-staining layer of osteoid tissue (Fig. 9). In isolated regions such polyhedral osteoblasts are observed enclosed in part by osteoid matrix and in part by connective tissue of the marrow space.

At the periphery of one portion of the mass a thin strip of dense, markedly cellular, collagenous connective tissue appears to have been detached from the surface. Contained within this strip of connective tissue are many discrete small sections of bone. The matrix in the central portion of each of these bony

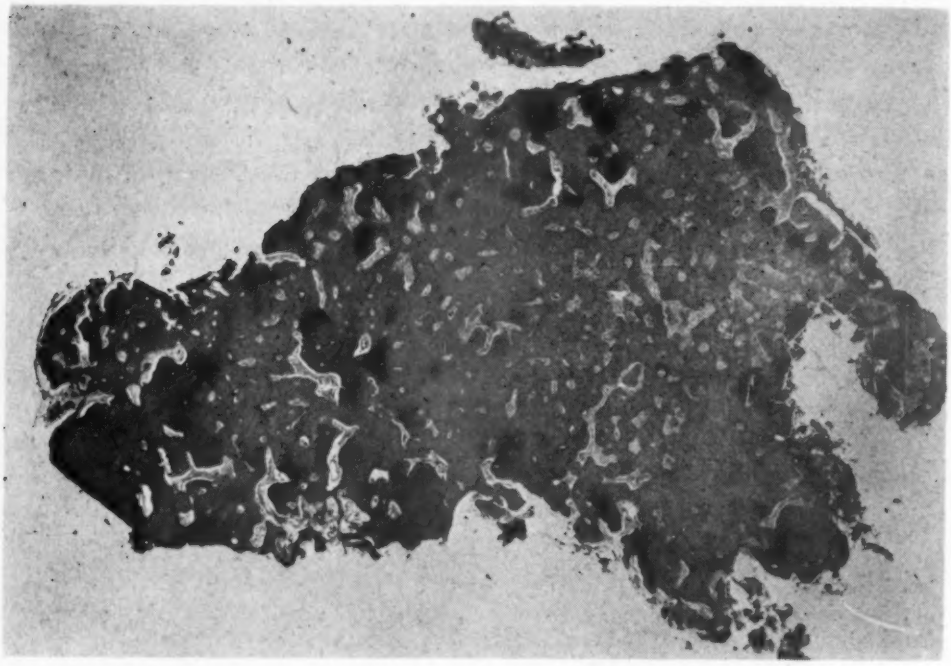


Fig. 8.—Low-power view showing closely arranged thickened bone trabeculae and comparatively small marrow spaces. Note connective tissue mass apparently detached from upper surface. (Hematoxylin and eosin, ×13.)

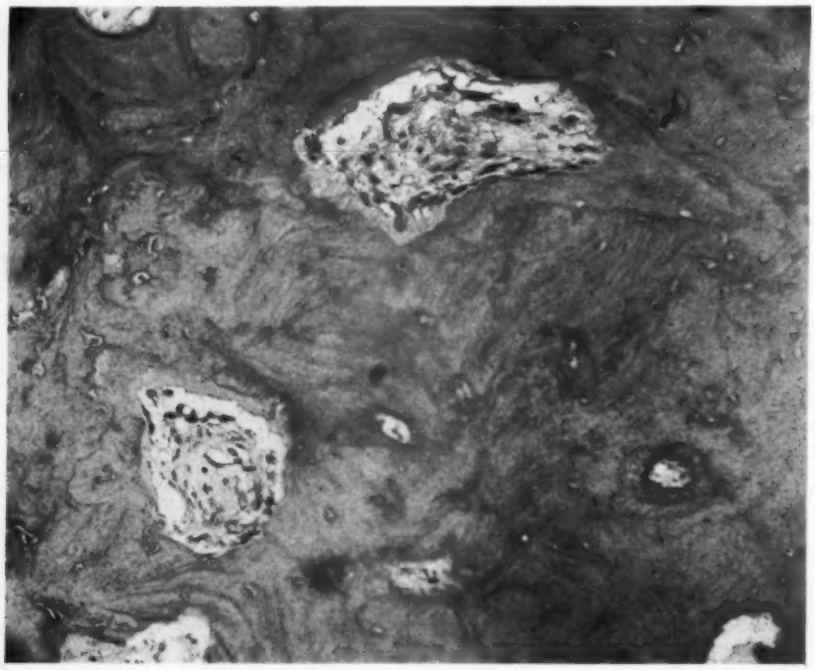


Fig. 9.—Detail of Fig. 8, showing osteoblasts adjacent to pale-pink osteoid matrix along endosteal margins of marrow spaces. Note irregular curvilinear markings in bone matrix. (Hematoxylin and eosin, ×200.)

sections is darkly staining and contains an occasional irregularly shaped osteocyte lacuna and enclosed osteocyte. The periphery of these bony sections is formed by a comparatively thin, pale-staining matrix in apposition with the outer border of which a beadlike layer of osteoblasts is seen. (Fig. 10.)

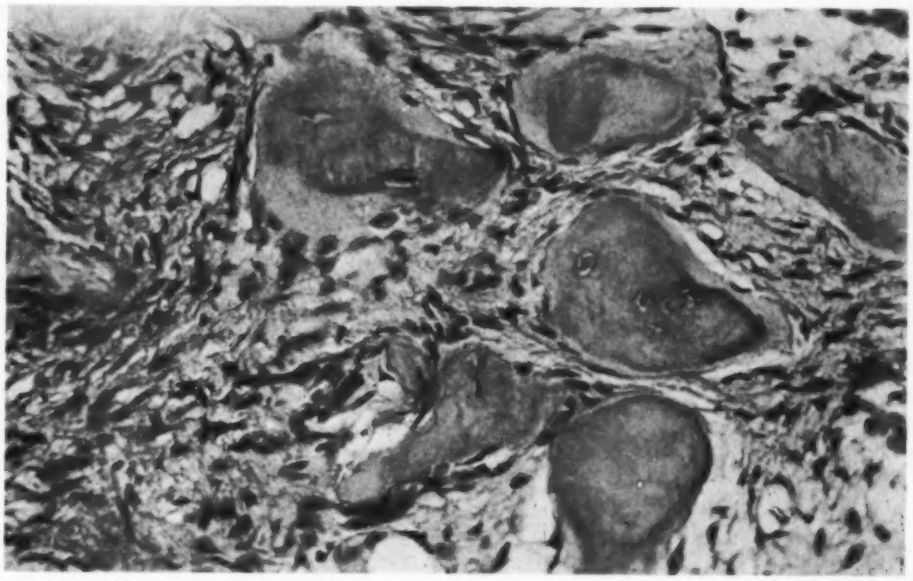


Fig. 10.—Detail of Fig. 8, showing bony inclusions within connective tissue detached from surface of lesion. Note pale-staining osteoid matrix around periphery of bone segments. (Hematoxylin and eosin, ×420.)

In essence, the microscopic picture of this lesion is one of bone condensation. The thickened bone trabeculae and marrow spaces which are diminished in size are responsible for the pronounced radiodensity which is far greater than that produced by the trabecular pattern which is normal for the cancellous bone in the mandible. The curvilinear markings in the bone matrix, noted above, are indicative of previously existent, irregularly arranged, successive zones of bone deposition. In addition, osteoblasts in apposition with pale pink osteoid matrix characteristic of new bone formation are seen along the endosteal bone surfaces and within the connective tissue capsule at the periphery.

COMMENT

When considered independently, the lesion responsible for the radiolucent radiographic area may be regarded as a localized osteitis fibrosa confined within a cystic cavity. Traumatic origin is not inconsistent with such a metaplastic change. On the basis of the microscopic findings, the dense osseous lesion productive of the radiopaque radiographic image is apparently either a circumscribed endostosis or central osteoma of the mandible.

When examined together, these lesions appear as diametrically opposed stages of a basically similar metaplastic equilibrium. In the former instance, the change to connective tissue produces an osteoporosis; in the latter lesion the

formation of bone and the replacement of marrow contents result in osteosclerosis. The presence of some degree of osteoblastic activity in the osteoporotic lesion suggests the possibility of a metaplastic equilibrium which when altered in opposing directions could produce lesions such as those described above.

Because areas of altered radiographic appearance occurred in many areas in both the mandible and maxilla, the possibility of a generalized skeletal disturbance or systemic disease with manifestations in the jaws should be considered here. The normal laboratory findings, the absence of radiographically detectable changes in the tibia, fibula, radius, ulna, and skull, the general case history and physical findings, and the microscopic findings point to no recognized generalized skeletal disturbance or systemic condition capable of producing the lesions considered here.

CONCLUSION

A correlated case study has been presented in which the general history, and radiographic, laboratory, and microscopic findings were correlated in a patient with unusual osseous lesions in the mandible and in the maxilla. A definitive diagnosis could not be evolved from all the available information. A diagnosis of localized osteodystrophy of the mandible and maxilla is suggested.

III. CONSERVATIVE MANAGEMENT OF A LARGE CYST IN THE MANDIBLE

Case Report

Joseph P. Lazansky, M.D., D.D.S., D.M.D., and
Arthur H. Wuehrmann, D.M.D.

A 19-YEAR-OLD man presented himself for treatment at Tufts College Dental School on June 3, 1945.

The past history was essentially negative. The first symptom of his present disorder was the appearance of a "gumboil" in the lower right molar area, sometime in May of 1945. At this time a local practitioner incised the swelling and irrigated the area daily for about two weeks. During this period a tooth anterior to the area was extracted on the premise that it was responsible for the pathologic process. At the end of two weeks the patient was referred to Tufts College Dental School for diagnosis and treatment.

The clinical examination showed the patient to be of robust stature and in excellent health. There was a slight asymmetry of his face, the right side from the corner of the mouth as far as the angle of the mandible being slightly enlarged. The patient was not in pain and external pressure did not elicit any signs of acute inflammation. The intraoral inspection revealed an opening ¾ inch in length on the alveolar crest distal to the second premolar. The lower right first and second molars were absent, the third molar was apparently missing or unerupted. Through the small opening a large cavity containing pus and lined with a necrotic membrane could be seen.

The x-ray examination included lateral jaw films. These showed a large radiolucent area, sharply defined, and regular in outline, extending from the lower right second premolar region into the coronoid process of the mandible and involving the whole width and height of the body and ramus of the mandible. The compact bone everywhere was reduced to a shell of only a few millimeters thickness. High up in the ascending ramus of the mandible was an inverted third molar. (Fig. 1.)

The preoperative diagnosis was dentigerous cyst with secondary infection.

Treatment.—Since the cyst was infected, it was irrigated daily with hot saline. After enlargement of the old incision the wound was kept open with iodoform gauze dressings carefully packed against the circumference of the opening, on the alveolar crest. Two weeks later the accessibility of the cyst was sufficient to permit inspection. A biopsy was taken to exclude the possibility of an ameloblastoma and the displaced third molar was probed. The lining of the cyst appeared smooth, shiny, and clean.

At this time an impression of the entire lower jaw was taken with colloid material, and a lingual bar with cast clasps was constructed. With the lingual bar in position an impression of the opening of the cyst was taken and an obturator of acrylic processed to the metal skeleton.

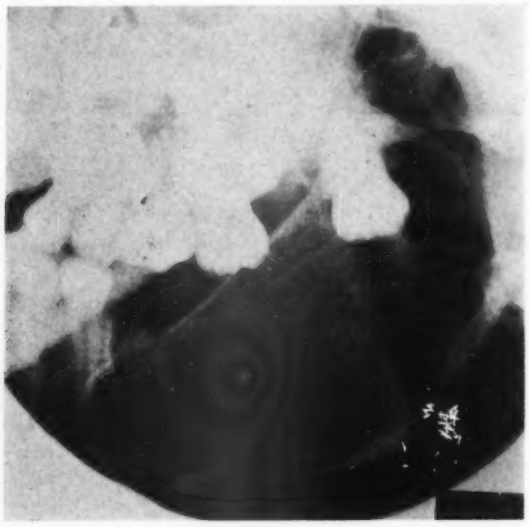


Fig. 1.



Fig. 2.



Fig. 3.

The obturator was inserted on July 15, 1945 (Fig. 2), and the patient was re-examined for sore spots for two more days. The patient was dismissed with instructions to wear the obturator continuously and to irrigate the cavity once or twice daily.

It was hoped that by maintaining an opening the cystic pressure would be released, permitting new bone formation similar to that observed in cases operated upon according to the Partsch method. Furthermore, it was felt that the renewed eruption of the displaced third molar might permit it to descend to a position where it could be more easily extracted.

In December, 1945, the patient returned for observation. In the interim, the obturator had not caused discomfort, and acclimation to the appliance was similar to that experienced by patients wearing partial dentures. At this time the cyst cavity was clean, covered with a smooth membrane, and devoid of all inflammation (Fig. 3). A few loose particles, probably food, could be removed by means of routine irrigation. On probing for the third molar the tooth appeared to have moved downward.

On Dec. 4, 1945, the x-ray examination showed a marked reduction in the size of the cyst as a result of new bone deposition. This was evident especially in the region distal to the second premolar. The cortical portion of the mandibular bone in the affected area was greatly increased in thickness. The third molar showed a change of position, being downward and forward as compared with the original x-ray (Fig. 4).

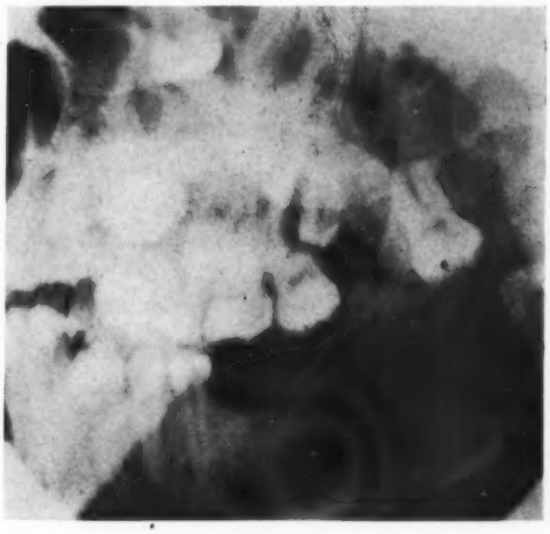


Fig. 4.

Comment.—The treatment plan in this case was considerably influenced by the weakness of supporting bone. Any extensive operation necessitating the removal of more bone, i.e., complete enucleation or even a typical Partsch operation, had to be avoided because of the possibility of a fracture. A mishap of this nature would cause complicated treatment especially since there were no teeth in the area for an internal fixation and there was insufficient bone for routine external fixation or wiring. We were also aware that enucleation of the cyst would denude a large area of bone and so expose it to further infection such as osteomyelitis with all the sequelae. Finally, complete removal was also a technical impossibility.

Among the many advantages of our treatment with the obturator, a few may be listed: (1) The patient did not have to undergo any major operation; (2) the danger of fracture and osteomyelitis was avoided; (3) the obturator prevented the entrance to the cyst from closing, and protected the cavity from food retention; and (4) the patient avoided numerous interim treatments.

There are several possible disadvantages in the use of obturators in cases of this nature. The resultant pressure and friction may cause denuding of primarily covered apices of neighboring teeth, and decubital ulcers at the orifice of the cavity. Likewise it is possible that overextention may prevent new bone formation on the floor of the cyst. However, these objections are not too important because all of these three complications can be avoided if the obturator is properly designed and relieved.

Finally, it should be borne in mind that available evidence supports the view that our treatment probably facilitates the eruption of the involved tooth. In this connection it has been shown that when dentigerous cysts are operated by the Partsch method, eradication of the cystic cavity proceeds much more rapidly when the tooth is left in position than when it has been extracted during the operation.

It is expected that because of the favorable movement of the third molar, an uncomplicated removal can be effected at a later date.

IV. COMPOUND COMPOSITE ODONTOMA

Case Report and Histologic Study

Irving Glickman, B.S., D.M.D., and Arthur H. Wuehrmann, D.M.D.

THE literature contains many references[1-7] to the occurrence, classification, and pathogenesis of calcified odontomas. Because detailed histopathologic studies are not commonly correlated with the gross and radiographic appearance of such lesions, the following case report of a compound composite odontoma is presented.

Radiographic Appearance.—Intraoral radiographic survey of a 14-year-old girl revealed an unerupted maxillary right cuspid, the crown of which was surrounded by a sharply demarcated, radiolucent zone. Superimposed upon the image of the crown were three discrete, relatively radiopaque masses, each mass measuring approximately 2 to 3 mm. in diameter. (Fig. 1.)

The cuspid and attached mass were removed and submitted for microscopic examination.

Gross Description.—Specimen consists of a normal-appearing maxillary cuspid the crown of which is enclosed within a grayish, glistening capsular membrane. On the lingual aspect of the crown, firmly adherent to the capsule, is a brownish, hard, smooth, nodular mass across the center of which is a slightly indented, horizontal depression (Fig. 2). Continuous with the lateral aspect of this knoblike mass is a small, firm, spherical protuberance.

Postoperative Diagnosis.—Cuspid with attached calcified odontoma. Specimen was embedded in celloidin previous to decalcification with an equal mixture of sodium citrate and formic acid, and sectioned for microscopic study.

Microscopic Description.—Longitudinal sections through the specimen reveal a normal-appearing cuspid, the enamel of which has been removed by decalcification in the course of preparation for microscopic study. At the periphery of the space formerly occupied by the enamel of the cuspid, a strand-like epithelial structure comprised of flattened, elongated, stratified, squamous epithelial cells and varying in thickness from 2 to 7 cells forms a continuous peripheral outline. In some portions this epithelial strand appears as a thin, hyaline, linear cuticular band.

External to the epithelial strand is a collagenous connective tissue capsule which encloses the epithelium at the periphery of the enamel space and is continuous with the periodontal membrane of the root.

On the lingual aspect of the coronal portion of the cuspid enclosed within the connective tissue capsule is a tumor mass comprised of the following dental tissues: enamel, dentine, pulp, cementum (Fig. 3). The tumor can be subdivided into two areas, each of which contains a portion of pulp, dentine, and



Fig. 1.—Radiograph showing embedded cuspid with discrete, small radiopaque masses superimposed upon the image of the crown.



Fig. 2.—Gross specimen showing nodular mass attached to capsule on lingual aspect of the crown.

enamel. For the most part, the pulp, dentine, and enamel are arranged so as to resemble two individual, dwarfed tooth crowns.

The following general findings apply to the individual tissues.

Enamel is deposited in relation to dentine and in many areas fills in lacuna-like depressions within the dentinal mass. In one region the enamel adjacent to the dentine has been completely removed in the course of decalcification. In general, however, bluish-staining masses of enamel remnants are scattered throughout the lesion. These masses on cross section present hexagonal prismatic outlines and in longitudinal section appear as parallel columns separated by linear, deeply staining threads (Fig. 4). The outline of enamel rods and interrod spaces are clearly defined in this mass.

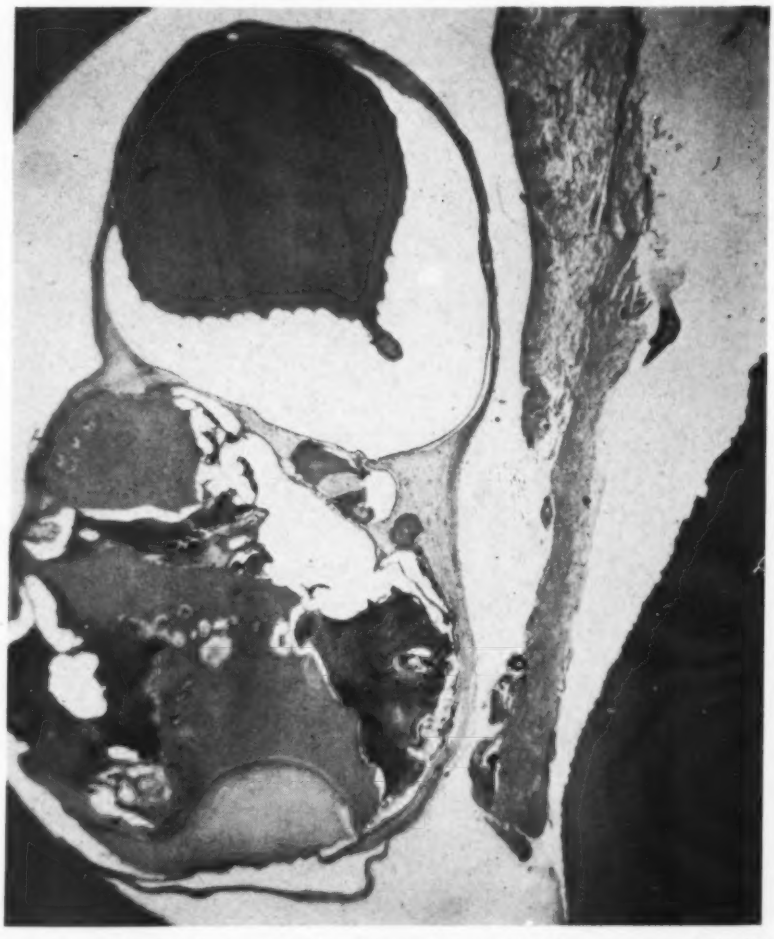


Fig. 3.—Low-power section showing dental tissues arranged to form two individual dwarfed crownlike masses on lingual surface of cuspid. (Hematoxylin and eosin, ×40.)

The *enamel epithelium* outlining the masses of enamel found throughout the section presents a variety of appearances. In some areas it is composed of flattened, cuboidal cells 3 or 4 layers in thickness, along the inner aspect of which is a thin, pale-stained hyaline strand. In other areas the epithelium

presents the appearance of a reticulum of stellate-shaped cells, along the inner aspect of which columnar ameloblasts with large spherical nuclei and clearly staining chromatin granules are seen (Fig. 5). These cells can be clearly demarcated from each other and appear to lie upon a thin, hyaline-staining basement membrane. Within the stellate reticulum, many of the cells are enlarged, the cellular membranes ruptured, the nuclei shrunken and hyperchromic with resultant vesicle formation. In a few areas the enamel epithelium is comprised of 15 or 16 flattened, spherical cells, within which appear discrete cystlike spaces enclosing a deep bluish-staining, amorphous concretion (Fig. 4).

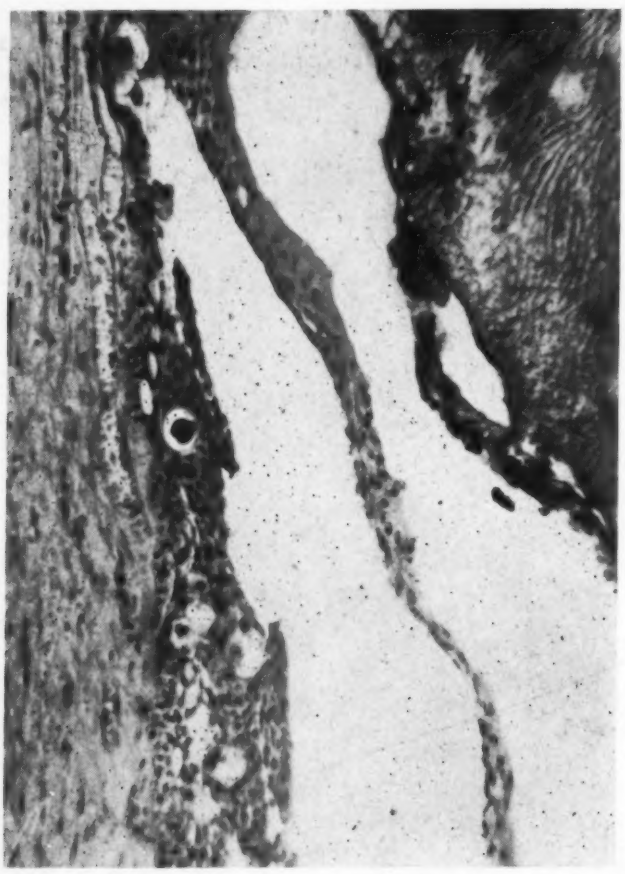


Fig. 4.—Enamel remnant and enamel epithelium with encapsulated small enamel pearls. (Hematoxylin and eosin, ×420.)

Large masses of *dentine* with clear-cut tubular outlines continuous with the odontoblastic layer of the pulp are seen adjacent to the enamel and lying between it and the pulp. The tubules pursue a relatively straight course (Fig. 6). Immediately adjacent to the odontoblastic layer of the pulp is a pale-staining, clearly demarcated zone of predentine. Within the predentine zone can be seen spherical, deeper-staining globular areas of calcification (Fig. 7). The continuity of the dentinal tubules in many areas is interrupted by irregularly outlined spaces within which the enamel remnants described above and enamel epithelium are seen.

The *pulp* is contained within a semicircular space, the arc of which is outlined by the dentine and the flattened portion of which is enclosed within the fibrous connective tissue capsule which surrounds the entire tumor (Fig. 6). The pulp is comprised of a dense meshwork of stellate-shaped connective tissue cells interspersed in a richly collagenous fibrillar matrix. At the periphery of the pulp immediately beneath the dentine is a layer of deeper-staining spherical cells, 7 to 9 cells deep, which comprises the odontoblastic layer. The pulp is relatively avascular throughout and presents only occasional capillaries.



Fig. 5.—Enamel epithelium showing columnar ameloblasts. Enamel remnant visible at bottom of section. (Hematoxylin and eosin, ×420.)

In one region a concentric mass is seen within the pulp in close relation to the dentinal layer. This mass presents a laminar appearance outlined by deep-staining concentric, thin, linear demarcations (Fig. 8). The entire substance of the mass is comprised of a pink, coarsely staining substance through which irregularly outlined, abruptly terminating, slightly tortuous tubular spaces can be seen at right angles to the concentric laminations. A thin, paler-staining area forms the outermost portion of this mass. In apposition with this paler-staining area is a discontinuous, beadlike periphery of spherical, pulpal cells.

The *cementum* occupies the smallest portion of the specimen and appears within the peripheral connective tissue capsule as an isolated globule in relation to enamel epithelium at the periphery of two zones of enamel (Fig. 9). The cementum is amorphous, acellular, and deeply staining and presents a minutely indented linear periphery, a small portion of which is filled in by a pale-staining amorphous substance (Fig. 10).

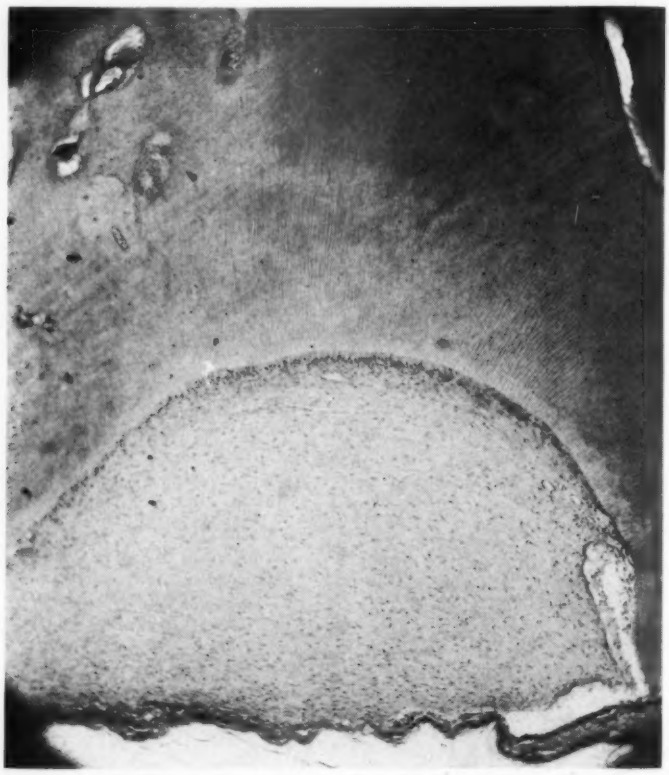


Fig. 6.—Dentine and pulp, showing tubular nature of dentine and odontoblastic layer of pulp. (Hematoxylin and eosin, ×110.)

Comment.—This specimen consists of a cuspid which is essentially normal; the crown is enclosed within a connective tissue capsule which is continuous with the periodontal membrane. Enclosed within this connective tissue capsule on the lingual aspect in the coronal portion of the tooth is an encapsulated tumor composed of dental tissues. This tumor contains enamel, dentine, pulp, and cementum. The cementum appears as an isolated mass. The other tissues are arraged so as to form two individual dwarfed dental crowns. A diagnosis of *compound composite odontoma* may be made on the basis of the microscopic findings.

For the most part, the tissues within the tumor present the microscopic detail of normal adult dental tissues. There is no evidence of malignancy or inflammatory change. The tumor is completely encapsulated by connective tissue.

The following microscopic features are worthy of note:

a. The enamel epithelium presents a variety of appearances and in some regions small enamel pearls.

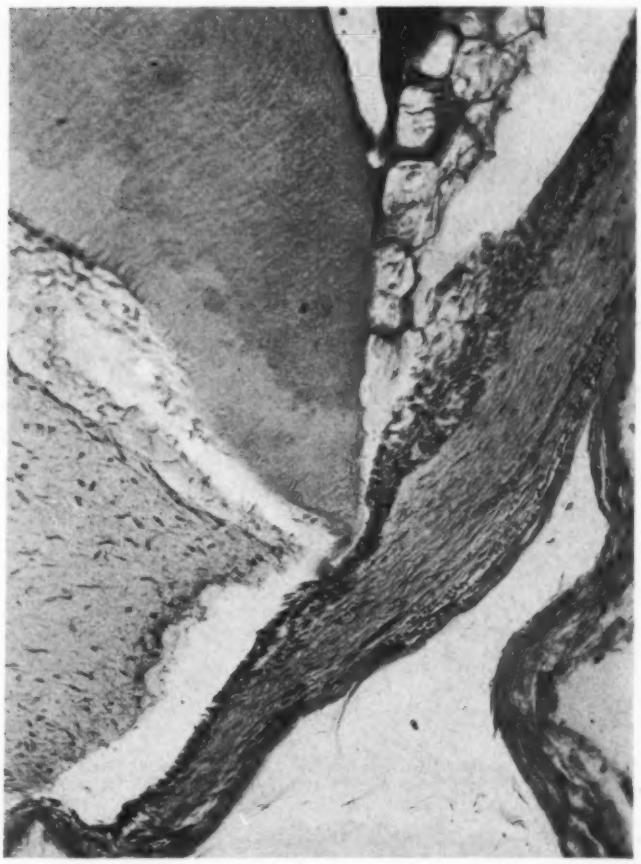


Fig. 7.—Detail of Fig. 6, showing calcification globules at margin of predentine zone. Note relation of dentine, enamel remnant, and enamel epithelium at termination of enamel on dentine. (Hematoxylin and eosin, ×220.)

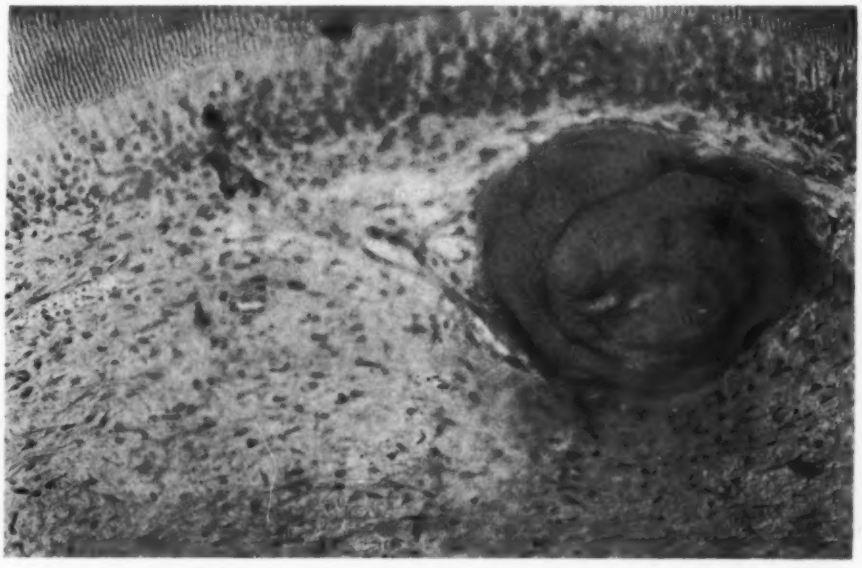


Fig. 8.—Spherical laminar mass in pulp tissue. (Hematoxylin and eosin, ×420.)



Fig. 9.—Spherical nodule of cementum enclosed within connective tissue projection from external capsule. (Hematoxylin and eosin, ×95.)

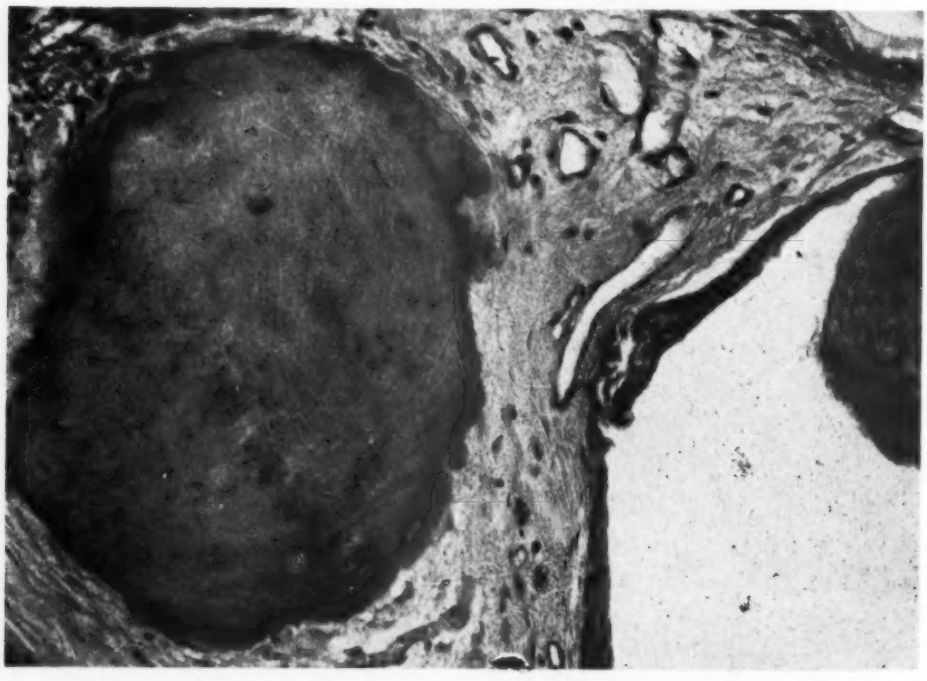


Fig. 10.—Detail of Fig. 9, showing a cellular nature and irregularly indented margin of cementum mass. (Hematoxylin and eosin, ×410.)

b. A large portion of the enamel was acid resistant and remained even after the decalcifying process to which this specimen was subjected in the course of preparation. The persistence of the enamel in the tumor to such a degree that the rod and interrod spaces could be clearly discerned was in marked contrast to the more or less complete dissolution of the enamel of the normal cuspid to which the tumor was adherent. The relative insolubility of a large portion of the enamel in the tumor may be assumed to signify a relative immaturity of this substance in comparison with its condition in the normal tooth. This assumption is based on the concept that increasing over-all solubility of enamel to decalcification is associated with progressive maturation.

c. In one area the pulp contained a true denticle which consisted of laminar, relatively atubular, irregular dentine. In a tumor such as this, ectopic dentinogenesis productive of such a structure is consistent with the over-all picture of anomalous development.

d. The relation of odontoblasts to the predentine zone and to the regularly tubular dentinal structure was essentially normal.

e. The absence of any attempted root formation was a notable finding. The only evidence of cementum was limited to a small isolated mass which occurred in an unusual position separated from, but adjacent to, two zones of enamel.

Conclusion.—This is a case of a calcified dental tumor contained within a connective tissue sac on the lingual aspect of the crown of an embedded cuspid. The tumor is a nonmalignant, compound composite odontoma.

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V. FAILURE OF UNION IN A MANDIBULAR FRACTURE AND ITS SUCCESSFUL NONSURGICAL TREATMENT

JOSEPH P. LAZANSKY, M.D., D.D.S., D.M.D., AND
ARTHUR H. WUEHRMANN, D.M.D.

A 24-YEAR-OLD dental student presented himself for treatment at Tufts College Dental School on Sept. 12, 1945. The past history revealed that the patient had suffered injuries to his face and jaw on July 21, 1945. First-aid treatment consisted of cold pack applications and hypodermic injection of analgesics. On July 23, the patient was transferred to a second hospital where x-rays were taken and a double fracture of the mandible was detected. Intermaxillary wiring was applied and the patient was injected with 30,000 Oxford units of penicillin every three hours for an indefinite period. After discharge from the Army, the patient enrolled as a student at Tufts College Dental School.

Our initial examination found the patient to be in good health although underweight, and complaining of difficulty in obtaining proper nourishment because the intermaxillary wires were still in position and it was necessary to exist on semiliquid food.

On external inspection, the patient's face failed to show any abnormality or asymmetry. Intraoral examination revealed that the upper and lower jaws were wired together according to Gilmer's method. The gingiva was highly inflamed due to irritation, but no signs of infection of the bone in the fracture lines could be seen.

X-ray examination revealed two fractures, one in the angle of the left mandible, the other between the lower right cuspid and lateral incisor. The former fracture included the socket of the third molar which was still in position. In this region bone repair could be detected. The fracture between the lower right cuspid and the lateral did not show any regeneration in spite of the fact that the fragments were in good apposition (Fig. 1). On Sept. 12, 1945, because of an acute gingivitis, it was necessary to remove the wires from the teeth in the maxilla and the mandible. Digital examination revealed excellent union of the posterior fracture but the anterior fracture was not yet consolidated and the fragments were movable to the extent of about 3 to 4 mm. in all directions.

Since the alignment of both fractures was excellent, the jaws were left unwired for the next several days so that the gingiva might recuperate. In the interim, the patient was advised to consume only soft food. Since the fracture had occurred two months previously, it was felt necessary to determine the factor responsible for the nonunion of the anterior fracture.

However, laboratory evidence (Wassermann, blood calcium, phosphorus, phosphatase, vitamin C level) was essentially negative. Nor was it possible to find any local factors interfering with the bony union.

The two teeth adjoining the anterior fracture line were tested for vitality and found vital. At this time it was felt advisable to make a decision as to the future treatment. Two possibilities existed: one, surgical interference, the other, conservative refixation. The patient was very cooperative and requested the latter treatment so as not to be held up in his studies. Accordingly, another attempt of fixation was made. A split acrylic splint was constructed for the lower teeth. This gave the patient the opporunity to eat soft food. This activity resulted in an improvement of the circulation in the damaged area. The splint was constructed in such a way as to make normal occlusion and masticatory movements possible, every part of the splint being ground completely out of occlusion.

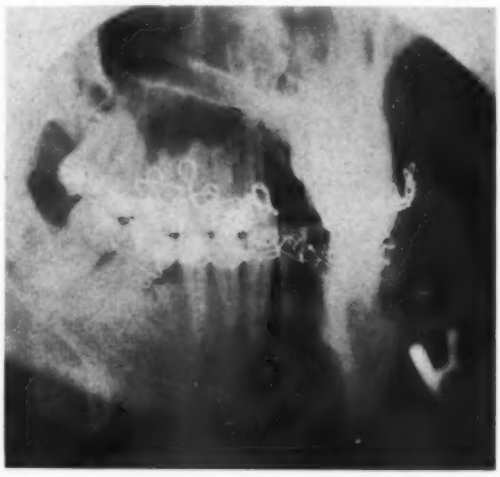


Fig. 1.

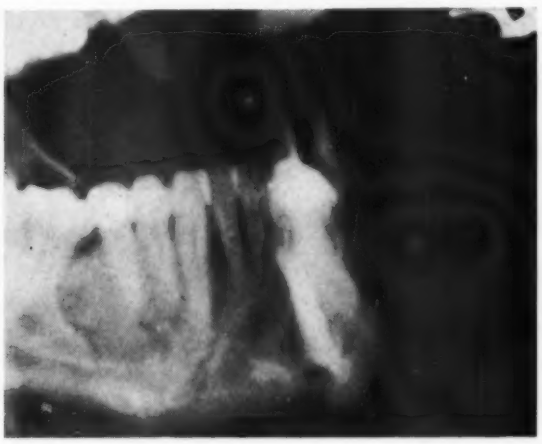


Fig. 2.

The splint was inserted on Oct. 1, 1945. During the first week the patient gained 5 pounds and his general condition subjectively was much improved.

The splint was carefully removed weekly, the oral cavity irrigated and the splint reinserted. On October 14 the fracture showed only slight mobility. On December 13 the splint was removed, and since no mobility could be detected, it was not reinserted. The patient was advised to chew softer foods for the next three weeks.

X-rays were taken on Jan. 12, 1946, and showed partial obliteration of the anterior fracture line (Fig. 2).

VI. DECALCIFICATION OF TEETH IN CASES OF ACHLORHYDRIA

HELMUT A. ZANDER, D.M.D., D.D.S., M.S.

CASE 1.—Mr. F., aged 40 years, was referred to Tufts Dental School Clinic for diagnosis and treatment planning for his upper six anterior teeth. About nine months before the patient had noticed a progressive wear on the lingual surface of these teeth.

When questioned, the patient stated that for the past four years he had suffered from a condition characterized by a deficiency in gastric hydrochloric acid. To remedy the situation he had been instructed to take dilute hydrochloric acid three times daily. After the practice had been followed for two years, it was discontinued, only to be resumed a year before he was seen at this Clinic.



Fig. 1.—Roentgenogram of upper incisors of Case 1. The incisal edges are irregular and appear radiolucent.

Examination of his mouth reveals a smooth, hard, shiny, and worn lingual surface of the upper incisors and cuspids. (Figs. 1 and 2.) The incisal third of the central and lateral incisors consists only of the labial enamel layer which is so thin that it appears transparent. The incisal edges are fractured in an ir-

regular fashion. The labial surfaces of all his teeth are free from decalcification, abrasion, or erosion defects. The lingual surfaces of all teeth except the upper six anteriors are free from defects.

Diagnosis: Abnormal abrasion of lingual surface of upper anterior teeth due to hydrochloric acid.



Fig. 2.—Photograph of same case of achlorhydria as Fig. 1. The incisal third of the teeth is thin and transparent. The enamel of the lingual surfaces is missing.

Case 2.—Mrs. P., aged 55 years, presented herself at Tufts Dental School Clinic, March 20, 1945, for dental restorations. Upon examination the patient showed four extremely thin upper incisor teeth. The enamel of the labial surfaces was intact but so thin that it was transparent. The lingual surface of these teeth was devoid of enamel and most of the dentine. In the cingular area a dark brown spot could be seen. This spot represented irregular dentine at the previous site of the pulp chamber. None of her other teeth presented abnormalities.

The patient admitted to suffering from achlorhydria for the past twenty-four years. As part of the treatment during this time she had taken dilute hydrochloric acid before meals three times daily.

Diagnosis: Abnormal abrasion of lingual surface of upper anterior teeth due to hydrochloric acid.

COMMENTS

Partial achlorhydria is a fairly common condition. The time-honored treatment for the deficiency is dilute hydrochloric acid taken by mouth three times daily before meals. Patients are advised to take the acid through a glass tube which is placed far back in their mouth. Most of these patients protect all the lingual surfaces of their lower teeth and their upper posterior teeth with

their tongue. However, they find it impossible to guard the lingual surface of the upper anterior teeth and the result is an unsightly defect. The severity and extent of the defect vary according to the amount of wear of the lingual surface of the anterior teeth. The lesion is always hard, smooth and shiny, resembling the classical picture of erosion and is not comparable with the decalcification seen in caries. The affected teeth in Cases 1 and 2 were restored with porcelain jacket crowns. However, it should be possible for dentists to construct a simple removable appliance that would protect the upper anterior teeth during ingestion of hydrochloric acid. Such an appliance would prevent the unsightly defects and ultimate costly restoration of these teeth.

Since the condition bears a strong resemblance to erosion, it may offer a possible explanation of the mechanism of similar unexplained conditions.

VII. CALCIFIED EPITHELIAL CYST IN THE SUBCUTANEOUS BUCCAL TISSUE

Case Analysis

Irving Glickman, B.S., D.M.D., Edward J. Trafidlo, D.D.S., and Samuel Pruzansky, D.D.S.

CALCIFIED tumors occur infrequently in the subcutaneous tissue of the oral buccal mucosa. The following case of a calcified mass in this area in which it was possible to substantiate clinical and radiographic diagnoses by histologic examination is therefore reported.

Miss C. W., No. 429714, aged 30 years, reported to the Dental Department of the Boston Dispensary for routine care. The patient was blind due to an injury incurred at the age of 10 years. She also gave a history of chronic vesicular dermatitis of forearm, back, and neck since the age of 5 years.

Routine intraoral radiographic examination revealed a sharply outlined spherical, radiopaque mass, approximately ½ inch in diameter, in the region distal to the crown of the lower left second molar. Throughout the mass were irregularly shaped areas of varying radiolucence. The radiographic appearance of the lesion suggested a concentrically lamellated internal structure. (Fig. 1.)



Fig. 1.—Intraoral film, lower left molar area showing calcified spherical mass.

This mass had escaped detection by visual clinical examination because it was deeply embedded in heavy buccal pads of tissue and produced no alteration in either the surface appearance or contour. However, palpation disclosed a freely movable, hard, spherical mass in the subcutaneous buccal tissue opposite the region of the lower left third molar. The patient stated that the mass had reached its present size slowly since she first noticed it approximately eighteen

years before. The patient gave no history of pain in the region at any time but did note that she tended to pinch it between her teeth occasionally. All other opposing molars on that side were present.

Under novocain anesthesia, a spherical, hard, gray, encapsulated mass, 1½ cm. in diameter, was readily dissected from the surrounding tissue. The area was sutured and healed uneventfully.*

Differential Clinical Diagnosis.—Epithelial cyst, sebaceous cyst (wen), salivary calculus, ossifying fibroma, dermoid cyst, odontoma, or calcified tubercular lymph gland.

Microscopic Description.—Section consists of a spherical mass comprised of concentric amorphous lamellations at the periphery of which is a continuous, thinned-out, avascular, fibrosed, connective tissue capsule. (Figs. 2 and 3.) There is no evidence of malignancy.

Microscopic Diagnosis.—The appearance of this lesion is consistent with that of a calcified benign epithelial cyst, the epithelium of which has undergone atrophy.

Discussion.—On the basis of the microscopic findings, either of two possible mechanisms may have been responsible for this lesion: the invagination of stratified squamous epithelium from the oral mucosa into the underlying connective tissue as the result of trauma, with the subsequent formation of a cyst filled with sebaceous material; or the obstruction of an ectopic sebaceous gland with formation of a retention sebaceous cyst (wen). In either instance, calcification of the sebaceous contents and atrophy of the epithelial lining were subsequent occurrences responsible for the consistency and microscopic appearance of the lesion at time of removal.

Subcutaneous, firm, spherical cysts of varying size have been described in the literature, occurring on the palmar surface of the hands and fingers and on the feet.[1] The nomenclature applied to these cysts has not been very constant but it is generally agreed that they result from trauma. Because the cyst described in this report occurred in an area frequently subject to injury in the course of mastication, the probability of a traumatic origin followed by epithelial changes and calcification as described above is not unlikely and is consistent with opinions regarding similar cysts found elsewhere in the body.[2-5]

In considering the probable origin of the lesion presented here it is pertinent to note Franke's theory that cysts arise from embryonic cell rests which are stimulated by trauma to produce epithelial pearls, and later become cystic.[6]

The following experimental conditions under which the formation of epithelial cysts has been reported are of interest:

> Direct implantation of a section of skin during an injury. If epidermis alone is implanted a smooth cyst results, while for the production of papillae or accessory structures the whole thickness of the skin must be buried.[7, 8]

*Operation performed by Dr. Joseph P. Lazansky, Assistant Professor of Oral Surgery, Tufts Dental School.



Fig. 2.—Low-power section showing peripheral capsule and lamellated contents. (Hematoxylin and eosin, ×13.)

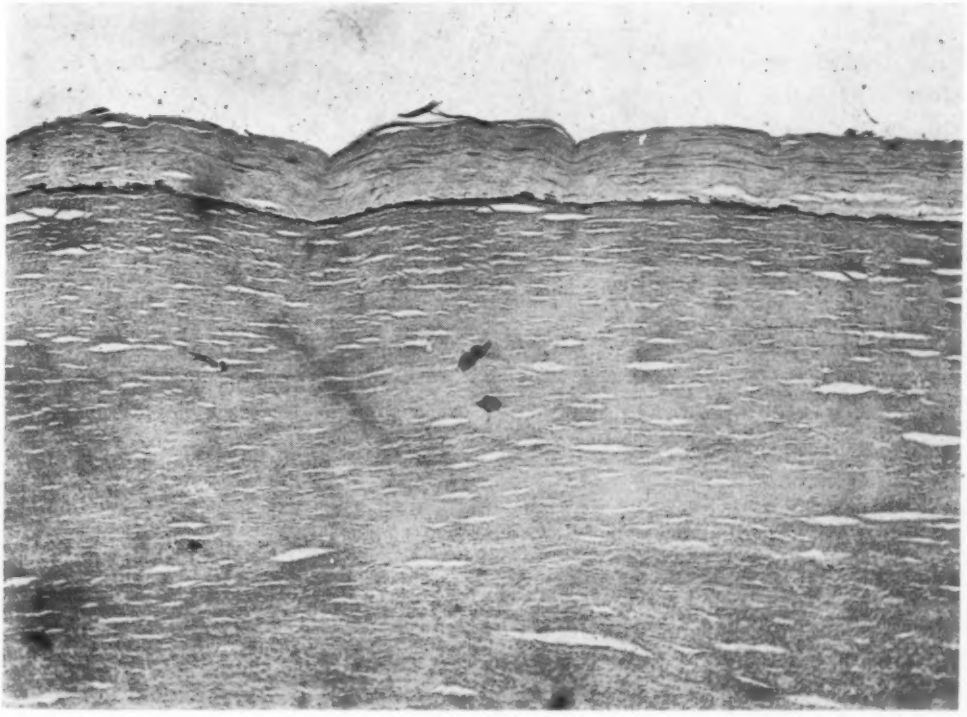


Fig. 3.—Detail of Fig. 2, showing fibrous capsule and granular nature of contents. (Hematoxylin and eosin, ×110.)

Injury by a foreign body which traumatizes sweat glands, sebaceous glands or hair follicles. Epithelium proliferates at the point of injury and grows to cover the implanted substance.[9]

Injury producing a foreign body reaction with granulomatous formation resulting in an epithelial cyst.[10]

Although it is likely that the cyst described here originated from proliferation of the oral stratified squamous epithelium secondary to trauma, its derivation from the obstruction of an ectopic sebaceous gland cannot be completely ruled out.

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Editorial

The Twenty-Seventh Annual Meeting of the American Society of Oral Surgeons

THE meeting of the American Society of Oral Surgeons was held at the Stevens Hotel in Chicago, on Friday and Saturday, Feb. 8 and 9, 1946. After greetings from the Local Arrangements Committee were extended to the members of the society by its chairman, Dr. Ralston I. Lewis, the president of the society, Dr. Sam H. Brock of Dallas, Texas, welcomed the members and guests. Among the latter was Dr. Ries Centeno of Buenos Aires, who recently published an excellent monograph entitled, *Cirugia Bucal.* Dr. Brock expressed the hope that the 1946 meeting would be the happiest and largest meeting ever held. He greeted in particular the members of the society returning from the military service, congratulating them on the contributions they have made while serving their country. These men, he stated, have played no small part in making our soldiers, sailors, and marines so physically fit that we were able to defeat our enemies much sooner than anticipated, and have done much to alleviate the suffering of the wounded, and have aided in the great task of restoring them to health.

"The Present Status of Oral Surgery as a Specialty" was the first paper of the Friday session. It was one of the outstanding contributions of the meeting, and a very timely topic. Dr. Douglas B. Parker of New York, the essayist, described first the historical development of oral surgery. He pointed out that many of the specialties were developed in times of war, and that during the Civil War several surgeons acquired great skill in jaw surgery. Among them was James E. Garretson, who is recognized as the father of oral surgery, and was the author of the first book on oral surgery. In 1869 he was appointed to the hospital of the University of Pennsylvania with the title of Oral Surgeon. While he and his contemporaries who followed, namely, Matthew W. Cryer, Truman W. Brophy, Thomas L. Gilmer, and John S. Marshall, had both medical and dental degrees or a medical degree only, there were other men with a dental degree alone who also took outstanding parts in the development of the profession. One of these was Chalmers J. Lyons, who, with great vision, trained many dentists to enter this neglected field.

The essayist pointed out that in the early part of the century oral surgery received a great boost by the general use of local anesthesia which made it possible for many men to undertake minor oral surgical procedures in their offices. Some men developed the specialty in hospitals, and those who were adequately trained received staff appointments.

Dr. Parker described how specialties developed similarly in medicine, and how specialty boards were set up to fix standards and to certify men properly

qualified to practice, and that today appointments in civil hospitals, government agencies, public health service, and veterans' hospitals are given only to those who are thus certified. He said that the time had come to set up without delay a National Board of Oral Surgery. He felt this board should have the backing of the American Society of Oral Surgeons, and stated that he had been unofficially told that the committee on specialty boards of the American College of Surgeons was willing to cooperate.

Dr. Parker also discussed what the preparation and training should be in oral surgery, a specialty which is recognized as the connecting link between medicine and dentistry. He stated that oral surgery today is taught only in dental schools, and that it is necessary for the oral surgeon to have dental training no matter what other training or degrees he may have. He also stressed the importance of additional study of the basic medical sciences to give the oral surgeon an adequate background for his work. For practical training, internships should be available in good teaching hospitals. He also stressed the fact that the field of oral surgery needs to be more clearly defined, and cited the list of oral surgical diseases approved in 1944 by the Surgical Executive Committee of the Massachusetts General Hospital.

The topic leader for the discussion was Dr. Leslie M. Fitzgerald of Dubuque, Iowa, who pointed out that the council of the society appointed a committee to organize an oral surgery board as long ago as 1938, but that during the war this committee felt that action should be deferred. Dr. Carl W. Waldron and Dr. Leroy M. S. Miner added to the reasons for the creation of a Board of Oral Surgery, and stressed the importance of setting it up without delay.

"The Effect of Elevating Periosteum From the Bone" by Dr. John F. Svoboda, was the second paper of the day. The presentation was based on a histologic study of the process of repair which follows the preparation of a mucoperiosteal flap in alveolectomy. The healing involves first a short stage of immediate reaction in the tissue, more or less of an inflammatory nature; second, a transformation stage in which osteoid is formed by the tissue; and third, a stage characterized by bone apposition. Healing takes about three weeks, but in the presence of pressure, such as might be exerted by a poorly fitting denture, resorption might set in and may continue throughout life.

"Oral Examination and Diagnosis" was the topic by Dr. Fred G. Heimlich of Indianapolis, Indiana. After discussing the various aspects of a thorough examination, Heimlich devoted a considerable part of his time to the discussion of the type of report that should be sent to the consultant who referred the patient. He recommended that it should consist of (1) a roentgen diagnosis, (2) data gathered from clinical examination and laboratory tests, (3) a diagnosis, and (4) recommendations regarding treatment.

"Surgical Management of Oral Carcinoma" by Dr. Fred A. Henny of the Ford Hospital in Detroit, Michigan, was an excellent presentation of today's best thoughts on the subject. We can endorse all that Dr. Henny said. The importance of a prompt diagnosis, although generally recognized, is often neglected; patients are treated by the general practitioner with mouthwash, silver

nitrate, or other harmful drugs, while valuable time is lost, and with it the chance for a good result. The importance of Broder's grading of carcinoma from the microscopic appearance is not always recognized, although it gives valuable information which may influence the selection of the method of therapy. He felt that of equal importance are clinical factors, the duration of the disease, the age and general health of the patient, and the presence or absence of metastases. Also, the type of growth should be investigated. He cited Blair who stated that fungating lesions have a better prognosis than the ingrowing or invading type.

After describing some of the so-called precancerous lesions, he discussed the lesions according to occurrence in the various regions of the oral cavity. For treatment of carcinoma of the lip he recommended excision and electrocoagulation. He felt that carcinoma of the buccal mucosa requires full thickness excision and closure of the skin by suture, or at least excision to the skin followed by electrocoagulation of the base and margins. The prognosis is poor when the tongue is involved by carcinoma; hemiglossectomy is advised with the application of radium into the posterior and median aspect of the excision wound. The prognosis is poorest in carcinoma of the floor of the mouth; Henny recommended irradiation or electrocoagulation. Cancer of the upper jaw has the best prognosis unless far advanced, with involvement of the paranasal sinuses; treatment is by coagulation. In carcinoma of the mandible, one must always keep in mind the possibility of invasion of the mandibular canal. Resection should always be anterior to the mental, and posterior to the mandibular, foramina. Metastases play an important part in the final result; lymph nodes may be involved even though they are not palpable. Henny favored neck dissection in most cases, with the exception of the slowly metastasizing grade 1.

The discussion was led by Dr. Sheldon L. Brockett of San Diego, California. He emphasized that patients should be referred promptly to a surgeon or roentgenologist familiar with cancer treatment. Other points brought out in the discussion were the importance of extraction of infected teeth to prevent x-ray necrosis of the jaw, and the advisability of extracting the upper and lower incisors to create adequate space for the use of an intraoral cone such as recommended by Dr. Hayes Martin for x-ray treatment of cancer of the floor of the mouth. It was pointed out that relief of pain is an important part of cancer treatment, particularly in hopeless cases. Drugs should be given first; later, alcohol injections may be used. They give relief for as long as six months. Nerve section helps in other instances. In evaluating surgery and irradiation, the point was made that irradiation may produce scar in which cancer cells remain, and that, if possible, excision should be favored, or a combination of all these methods: surgery, x-ray, and radium therapy.

"*How to Ruin an Oral Surgery Practice*" was a topic ably handled by Dr. W. T. Ewing of Akron, Ohio. Some of the statements and recommendations provided a relief from the more serious parts of the program. Since in a specialty one depends almost entirely on referred work, a good motto is to play fair with your contemporaries, treat them as you would like to be treated yourself. Patients should be made to understand that accidents or complications

that may have arisen might have occurred even if he had come to you in the first place. No one is so good that he is infallible, and no matter who the doctor is, the world will go on long after he has been forgotten.

Dr. Thomas Conner of Atlanta, Georgia, stated that all his life he had tried to build a good practice, and that it seemed strange to come and listen to Dr. Ewing to find out how to ruin it (applause).

"The Treatment of Fractures of the Mandible and Facial Bones," a moving picture demonstration by Dr. Karl Hayden Wood, concluded the afternoon session. By means of a sound film he demonstrated many operations for the reduction and fixation of face fractures. Dr. Frederic W. Coggan of Kalamazoo, Michigan, started the discussion, which developed like a snowball, since the subject was a very controversial one, and particularly since some of the demonstrated procedures were outdated. Dr. Wood brought out in his closing remarks the fact that some of the operations had been photographed years ago, which explained the discrepancies that had been criticized in the general discussion.

"A New Method of Applying a Plaster Helmet for Head Anchorage in the Treatment of Maxillofacial Injuries" by Dr. Sanford M. Moose of San Francisco, California, was the first paper of the evening session. The plaster helmet, the construction of which was demonstrated by means of a moving picture film, was recommended as an improvement over the well-known head cap described by Scogin. Its advantage is rigidity without causing contusions of the skin. The rigidity is accomplished by carefully molding the soft plaster over the mastoid processes and the occipital prominence. Injury to the skin of the forehead, which occurs so often, is prevented by a piece of orthopedic felt placed on the skin while the cap is being molded, and removed after the plaster has hardened, so that a space is formed under the edge of the helmet. Dr. Moose, who seems to be an experienced fisherman, apparently combines business with pleasure, as many of the attachments he uses for various types of craniomaxillary fixation and traction are taken from his tackle box.

"Impacted Teeth" was not just another moving picture demonstration; it was the best one ever produced. Dr. H. Newman Brownson of Hollywood, California, demonstrated his method of removing unusual impacted teeth, and displaced teeth with associated dentigerous cysts. He gave an expert demonstration in the use of the impactor, but stated later that he used a chisel and mallet or burs when he felt he could employ them to advantage.

Dr. V. H. Kazanjian's film, *"Surgical Preparation of the Mouth for Dentures,"* was shown as the final demonstration. It presented Kazanjian's method of ridge extension as used in both the upper and the lower jaw. A mucosal flap from the oral surface of the lip is prepared and attached to the outer surface of the bone after removing the muscle attachments, and in the upper jaw, the nasal spine. The edge is attached to the periosteum with No. 000 catgut sutures. A rubber tube is then inserted into the newly created labioalveolar sulcus and attached by means of through-and-through mattress sutures tied over gauze on the outside of the face. In one case, gauze was saturated with tincture of benzoin and placed into the sulcus instead, and held there by means of the patient's

dentures, to which a flange had been added to hold the gauze in place and prevent the tissue along the ridge from moving down.

Dr. C. McRimmon of Fort Worth, Texas, and Commander Daniel F. Lynch discussed the advantages and disadvantages of the method. One of the most important disadvantages was the fact that patients object to it. Dr. McRimmon pointed out that many cases are unsuitable because of the extreme atrophy of the bone. He recommended that the thickness of the mandible in vertical dimension be estimated, and if it was not adequate, you had better say to the patient, "So sorry, good-bye."

Commander Lynch pointed out that in many cases gingival hypertrophies complicate the picture, and that contraction is a real problem whether tubes or dentures are used. He recommended stent skin grafting as a better solution of the problem. A model in plaster is made of the mouth, and the desired sulcus carved. Then a denture or a splint is constructed. A deep incision is made in the sulcus as far back as the tuberosity or the retromolar triangle; the denture is sterilized in alcohol and inserted. Greenstick compound is added so that the denture fits exactly, leaving no spaces underneath. A skin graft is taken from the forearm—this is best done by a plastic surgeon—and sewed over the underpart of the denture. It is placed into the mouth so that it fits firmly into the sulcus. It must be fixed so firmly that no movement can take place. In the mandible this is done by circumferential wiring, and in the maxilla by means of a winged Kingsley splint. After seven to ten days, the splint is removed and cleaned and put back in the mouth. After another two days an impression for a new denture is taken. The stent is to be replaced and worn until the new denture is made; this will prevent contraction. The use of penicillin, given intramuscularly every three hours during this period, prevents infection and safeguards the healing of the flap.

On Saturday the meeting was continued. General clinics were given in the afternoon. Dr. Robert L. Borland of Los Angeles, California, Dr. Orlan K. Bullard of San Diego, California, Dr. Albert C. Hitzelberger of Utica, New York, Dr. Frank H. O'Halloran of Evansville, Indiana, Dr. Berto A. Olson of Hollywood, California, Dr. Glen J. Pell of Indianapolis, Indiana, Dr. Edward Reiter of Cleveland, Ohio, Dr. Stanley Rice of Beverly Hills, California, Dr. William B. Spotts of St. Louis, Missouri, Dr. J. J. Stetzer, Jr., of Philadelphia, Pennsylvania, Drs. Ben J. and John F. Svoboda of Los Angeles and Chicago, respectively, and Dr. Phillip E. Williams of Dallas, Texas, demonstrated various principles of, and aids in, oral surgical procedures, and the treatment of complications. The dental officers of the U. S. Training Center, Great Lakes, Illinois, demonstrated *"Surgical Mouth Preparation for Dental Prosthesis."*

Saturday morning was devoted to the reading of additional papers. Dr. Horace L. Cartee of Miami, Florida, talked on the subject, *"Taking an Inventory,"* and Dr. William E. Durbeck of San Antonio, Texas, spoke on *"Mandibular Osteomyelitis,"* reviewing the various methods of treatment, up to and including the use of penicillin. The topic leaders were Dr. Aubrey L. Martin of Seattle, Washington, and Dr. J. D. Sullivan of Toledo, Ohio.

"Evaluation of Methods of Mandibular Osteotomy" by Dr. Reed O. Dingman of Ann Arbor, Michigan, was, to your reporter and many others, the high light of the meeting, particularly as so many procedures for osteotomy and osteoectomy have been advocated to improve facial appearance and function of the teeth in prognathism and open-bite.

The two principal operations in use today are the osteotomy in the ascending rami between the mandibular notch and the mandibular foramen, and the osteoectomy in the horizontal rami usually performed in the first molar region or posterior to it. The advantages of the osteotomy in the rami are ease of execution, avoidance of injury to the mandibular nerve, elimination of oral contamination of the wound, and preservation of all the teeth. The disadvantages brought forth are danger of injury to important structures: the parotid gland, the internal maxillary artery and facial nerve, derangement of the muscles of mastication, danger of overriding of the fragments, prolonged fixation to prevent nonunion or malunion of the surgical fracture and possible open-bite. The advantages of osteoectomy in the horizontal rami are accessibility, easy control of fragments, no interference with muscles, no danger of resulting open-bite. Disadvantages are the sacrifice of a tooth at the osteoectomy site, and the compounding of the wound into the oral cavity with danger of infection. In addition, the mandibular nerve is cut, causing a temporary anesthesia in the lower lip.

The two-stage operation which was recommended by New and Erich in 1941 to prevent injury to the nerve has been improved by Dingman. The method he advocates at present is performed in two stages at different times. The first stage consists of the extraction of the teeth and of cutting the section superior to the mandibular canal. The bone section, however, is not removed until the mucosa has healed. This allows the second operation to be performed under strictly aseptic conditions from an external approach. By means of burs, chisels, or rongeurs the lower section is excised, and the upper section removed. This leaves the nerve intact, being tucked into a cavity made in the spongiosa. The osteotomy in the ascending rami is frequently unsuccessful or unsatisfactory, but the osteoectomy in the horizontal rami gives uniformly good results.

The formation and incorporation of a National Board of Oral Surgery was voted in the business session of the meeting, and seven men were appointed to serve on this board. Thus, oral surgery has behind it another milestone in its history; a new epoch approaches which will bring with it great developments and a new high standard of service to our Nation.

K. H. T.

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Original Articles

THE IMPACTED MANDIBULAR THIRD MOLAR

P. Philip Gross, D.D.S., F.I.C.A.,* Philadelphia, Pa.

Part I. Abnormalities, Etiology, Results

CREDIT for analyzing and solving the peculiar problems presented by the impacted mandibular third molar must be given to Dr. George B. Winter[1] of St. Louis. His work is devoted largely to the classification and technique for removal of this molar.

ABNORMALITIES

Let us first examine the ways in which the mandibular third molar may be abnormal:

1. Failure to erupt.
2. Absence from the dentition.
3. Impaction:
   a. Malposition.
   b. Misplacement.

1. *Failure to Erupt.*—Unerupted teeth are normal in size, in shape, and in placement in the arch. Although there is adequate room, they fail to erupt because they have lost their physiologic impulse to erupt.

2. *Absence From the Dentition.*—Approximately 20 per cent of our population lacks, congenitally, one or more of the third molars. Milo Hellman[2] states that the incidence of congenitally missing third molars is higher among females than among males. After examining 735 adult individuals, male and female, he found that 201, or 23 per cent, lacked, congenitally, one or more third molars; whereas, of 314 females, 110, or 35 per cent, lacked one or more third molars congentially. In some of these cases all four of the third molars were missing. In my series of 250 cases (mandibular third molars only):

Number lacking mandibular third molars	40
Number lacking one mandibular third molar	28
Number lacking both mandibular third molars	12

(The left mandibular third molar was missing more often than the right.)

Photographs by Harold A. Thomas, medical artist and photographer.

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3. *Impaction.*—The term impaction means the confinement of a tooth within the jaw in a false position, a position from which its eruption into a normal position is prevented by the impingement of other teeth or because of its peculiar position in the jaw. The mandibular third molar is more frequently impacted than any other tooth. Of the 250 patients I examined, 27 had impacted mandibular third molars: 15 of the 27 had one impaction; 12 of the 27 had two impactions. There were more impactions on the right than on the left.

An unerupted, malposed, or misplaced mandibular third molar is often referred to as an impacted mandibular third molar. No distinct classifications are made of the malposed, the misplaced, or the unerupted teeth.

a. *Malposition:* Teeth are malposed when the crowns have erupted into the mouth, but are abnormal in their occlusal relation to the adjacent teeth. The mandibular third molar, the crown of which is erupted and is buccally inclined, is really a malposed mandibular third molar rather than an impacted third molar. This type of third molar creates disturbances along the inner surface of the cheek.

b. *Misplacement:* The misplaced mandibular third molars are literally misplaced—that is, they are in the wrong place. They are found in the ascending ramus of the mandible. The term misplacement is used when teeth are found outside the range of those parts where teeth are normally found.

ETIOLOGY

Conceivably, a variety of pathologic conditions might bring about retarded eruption, misplacement, or impaction of a tooth. Most frequently, the final cause of impacted mandibular third molars is mechanical—a lack of space in the arch. The frequency of impaction of the mandibular third molar is explained by the fact that because the tooth is the last in the series, anything which tends to lessen the space provided for the molars may leave insufficient space for the mandibular third molar between the ramus and the second molar.

RESULTS

The impaction of the mandibular third molar is usually a menace to the patient, often creating a great deal of discomfort, disarranging the dental arch by mesial force, and sometimes being responsible for the loss of the second molar. The mandibular third molar is the cause of inflammatory and pathologic changes in the surrounding tissues and the cause of purely subjective symptoms of neuralgic character.

Brophy,[3] speaking of the impacted mandibular third molar, says: "A tooth in such a position may cause extreme discomfort by reason of pressure directly upon the third or mandibular branch of the fifth nerve and thus cause neuralgia. It may cause extensive swelling, involving the surrounding soft parts. Such teeth, however, frequently remain throughout life without causing any disturbance."

Brown[4] states that "Direct irritation of the inferior dental nerve from an impacted tooth is quite a common cause of tonic spasm of the masseter muscle, causing trismus."

Blair and Ivy[5] state that "An impacted mandibular third molar may cause pressure against the crown of the second molar and cause decay of that tooth, or itself become the seat of caries around the point of contact. It may cause pressure absorption of the root of the second molar. Exposure and devitalization of the pulp from these causes may give rise to neuralgia."

Part II. Clinical Examination

Examination of the Exposed Parts of the Crown.—The exposed part of the crown of an impacted mandibular third molar should be examined ocularly and digitally, with the aid of a probe.

Examination of the Gum Tissue Over and Around the Crown.—The gum tissue is examined to determine the most favorable point for an incision through which to gain access to the ossistructure and the tooth. It is important to choose the most favorable line of incision so that, after the operation is completed, the gum tissue will protect the socket and the second molar. During the examination, attention should be given to the possible presence of infection beneath the flap. Such infection is usually due to the packing of food beneath the flap. Infection of the gum tissue usually indicates a pathologic condition of the underlying ossistructure.

Examination of the Second Molar.—The degree of firmness of the second molar is important. The distal surface should be examined thoroughly for caries. Testing the second molar for vitality should be routine. If this procedure is not followed, it will be embarrassing to the operator to find, after he has extracted the third molar, that the second molar was the factor which produced the disturbance.

Examination of the Opposing Maxillary Third Molar.—The opposing maxillary third molar should be examined for infection. If infection exists, it is better to extract the upper third molar before operating on the lower, to prevent the spread of infection to the lower socket from the upper molar.

Examination Where Extraction Has Been Attempted.—Where attempted extraction has taken place, the operator must determine the amount of trauma produced upon the surrounding tissue and the second molar. Where the exolever technique has been applied unsuccessfully, the operator should assure himself that no fracture of the second molar or the mandible has taken place.

Examination of Patient's Mouth and General Physical Condition.—After the operator has made a survey of the impacted tooth and its surrounding structures, he should examine the patient's mouth for the purpose of determining whether there are any infected teeth more extensively involved than the impacted tooth to be extracted. It is important that the mouth be in a hygienic condition at the time of the operation. If, in the judgment of the operator, the physical condition of the patient indicates a consultation with the family physician, the operation should be postponed.

Part III. The Roentgenographic Examination

In August, 1896, Dr. Edmund C. Kells of New Orleans presented before the Southern Dental Society, at Asheville, North Carolina, the first dental x-ray clinic ever held. He presented skiagraphs on glass plates, taken with five to fifteen minutes' exposure time, showing the perfect outline of the roots of the teeth.

Kells[6] states: "Immediately upon the introduction of the x-ray in my practice, I naturally began to ray all impacted and missing third molars, whereby the faulty methods for the removal of the third molar were manifest and I devised the operation of cutting the crown in two and removing the tooth in sections."

Previous to the application of the roentgen ray in dentistry, only those impacted teeth evident upon oral examination could be dealt with. It was advocated about this time that the second molar should be removed to allow the third molar to erupt, rather than that the third molar be removed.

At that time, to remove the third molar, the Physick forceps and the cowhorn forceps were used. One beak was applied at the lingual surface, well beneath the tooth, and the other beak over the buccal plate; the tooth, together with the buccal and the lingual plates, was then delivered to the lingual side. This indeed was a brutal method.

Practical experience has demonstrated that the successful removal of the mandibular third molar, impacted or otherwise, depends upon a correct roentgenographic examination and clinical diagnosis, in conjunction with an operative technique applicable to the case at hand. So much depends upon the correct preoperative diagnosis of the position of the third molar that it is essential a roentgenographic study be made of every case.

In order to have uniform success in the removal of these teeth, a definite roentgenographic technique is of utmost importance. If the examination is to be of any service, the picture must be taken at the correct angulation, as it is possible to have a number of views of the same tooth and show it in different positions by different angulations.

The intraoral, or lingual, view is the usual method of procedure. With this method more detail is obtained with less distortion of the tooth and the surrounding tissues.

The extraoral method is used to include large areas, for the localization of the teeth not in the regions of the intraoral examination, for cases of trismus that prevent the insertion of the intraoral film, and for the diagnosis of pathologic areas around and beyond the tooth. From a diagnostic standpoint, as an aid in the removal of the mandibular third molar, the intraoral method serves the purpose better.

In making the intraoral, or lingual view, the technique as described in the standard textbook of roentgenography is followed. The patient is placed in the upright position with the side of the mandible containing the affected tooth at right angles to the cone of the machine at 5 degrees above the 0 degree. The head is tilted distally so that, when the jaw is opened, the occlusal surfaces of

the mandibular molars are parallel to the horizontal plane of the head of the machine.

The position of the film for making the intraoral roentgenograph for the vertical type of mandibular third molar is to have the anterior border of the film pack at the mesial surface of the first molar and the upper border of the film pack parallel to the occlusal surfaces of the first and second molars (Fig. 1).



Fig. 1.—Position of film pack for making the roentgenograph of the vertical type of mandibular third molar.



Fig. 2.—Position of film pack for making the roentgenograph of the angular and the horizontal types of mandibular third molars.

In making the roentgenograph of the horizontal and the angular types of the mandibular third molars, the anterior border of the film pack is placed at the center of the first molar, and the upper border of the film pack is placed parallel to the occlusal surfaces of the first and second molars. It is a good policy to place the film pack as far distally as the patient will permit, to obtain as much as possible of the ossistructure distal to the third molar. A few drops of novocain at the lingual nerve facilitates the raying of these areas. (Fig. 2.)

The occlusal view is taken where the crown of the mandibular third molar is not visible. By the occlusal method, the deflection of the tooth is determined before surgery is attempted. The film is placed upon the occlusal surfaces of the molars, with the tube side of the film down and the anterior border of the film pack at the mesial surface of the first molar or as far distally as possible. The patient's index finger is placed over the film, or the patient is permitted to close lightly upon the film pack. The rays are then passed toward the inferior border of the mandible with the head extended backward as far as possible and to the side, so that the rays may pass at right angles to the film pack. This type of view shows the approximate degree of the buccal or the lingual deflection of the mandibular third molar as compared with the second molar. (Fig. 3.)

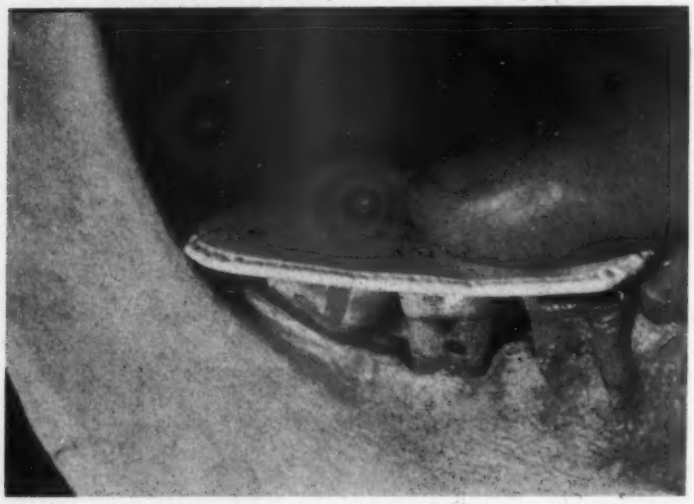


Fig. 3.—Position of film pack for making the occlusal roentgenograph of the mandibular third molar.

Part IV. Classification

The Winter classification of the mandibular third molar is based upon the following factors:

1. Position of the crown of the third molar.
2. Relation of the third molar to the second molar.
3. Character of the root formation.
4. Ossistructure surrounding the tooth.

The clinical examination of the contact points of the first and second mandibular molars will determine whether the roentgenograph has been properly made. The roentgenograph should show the teeth in the same position as they are in the mouth, with no overlapping of the contact points, if the first and second mandibular molars are in the normal position in the mouth. The enamel caps of the first and second mandibular molars should show clearly. In a properly taken roentgenograph there should be no loss of the enamel caps of the teeth. With the first and second mandibular molars having a normal

contact and the enamel caps showing clearly, an accurate diagnosis of the mandibular third molar can be made. (Fig. 4.)

1. POSITION OF THE CROWN OF THE THIRD MOLAR

The mandibular third molar is classified as follows according to the position of its crown (Fig. 5):

a. Vertical type.
b. Mesioangular type.
c. Distoangular type.
d. Horizontal type.



Fig. 4.—Lingual roentgenograph showing normal enamel caps of the first, second, and third mandibular molars. No overlap of the contact points of any of the teeth.

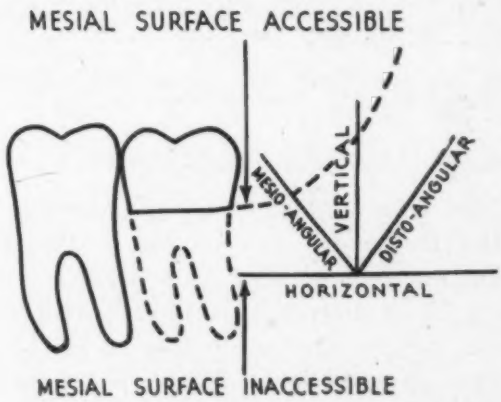


Fig. 5.—Classification of mandibular third molars.

Vertical Type.—In the vertical type the crowns of the third molars are parallel to the crown of the second molar. These teeth are normal mandibular third molars when fully erupted and in the normal position in the arch. (Fig. 6.)

Mesioangular Type.—Those teeth inclining anteriorly, or mesially, with the crown directed toward the second molar and the roots pointing toward the ramus (Fig. 7).

Distoangular Type.—Those teeth inclining distally with the crown directed toward the ramus and the roots toward the second molar (Fig. 8).

Horizontal Type.—Those teeth the crowns of which are at right angles to the crowns of the second molars (Fig. 9).

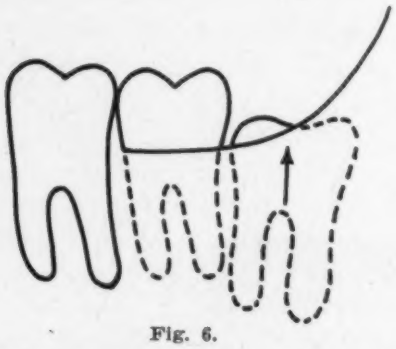


Fig. 6.

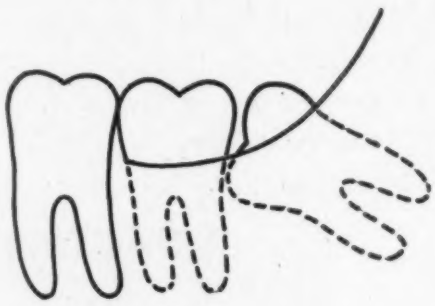


Fig. 7.

Fig. 6.—Vertical type mandibular third molar.
Fig. 7.—Mesioangular type mandibular third molar.



Fig. 8.

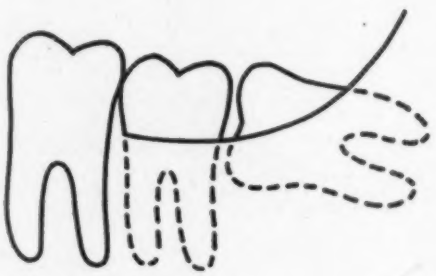


Fig. 9.

Fig. 8.—Distoangular type mandibular third molar.
Fig. 9.—Horizontal type mandibular third molar.

2. RELATION OF THE THIRD MOLAR TO THE SECOND MOLAR

As the second molar is an important factor to be considered in connection with the removal of the third molar, its retention in the arch, when in a normal condition, is of value to the patient, and every effort should be made to retain it. Caries may involve the occlusal and the distal surfaces of the second molar. A carious tooth is weak, and no dependence can be placed on the second molar where the tooth is carious or has a large filling. A conical-shaped second molar may easily pop out of its socket during the exolever movement. Pressure absorption of the distal surface of the distal root of the second molar leaves a very sensitive area after the removal of the third molar. Any pathologic condition involving the roots of the second molar will interfere with the healing of the socket of the third molar.

The position of the crown of the third molar as determined from the roentgenograph will be either vertical, mesioangular, distoangular, or horizontal. Each type may be in alignment with the second molar when comparing the

buccal and the lingual surfaces of the third molar with the corresponding surfaces of the second molar.

The crown of the third molar in any of the above-mentioned positions may also be deflected buccally or lingually out of alignment with the second molar; it may be deflected both buccally and lingually in what is termed bucco-lingual deflection, or it may be in a torsional position.

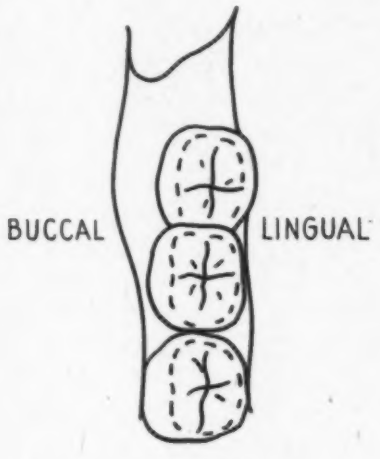


Fig. 10.

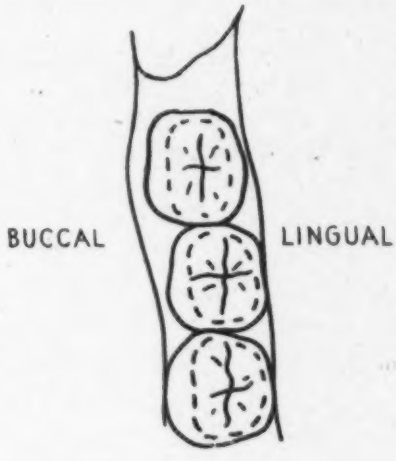


Fig. 11.

Fig. 10.—Occlusal view of alignment of the buccal and lingual surfaces of the mandibular second and third molars. In the lingual roentgenograph there would be no overlap of the contact points and no deflection of the third molar.

Fig. 11.—Occlusal view showing buccal deflection of the mandibular third molar.

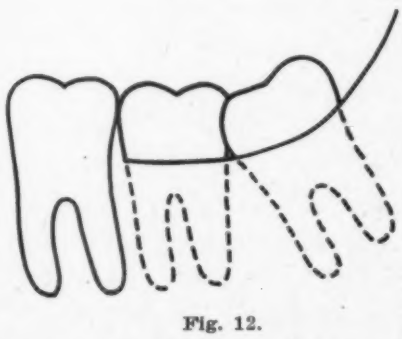


Fig. 12.

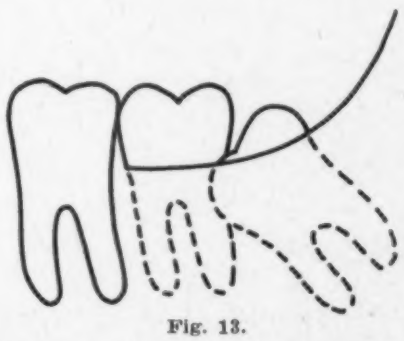


Fig. 13.

Fig. 12.—Lingual view. Normal contact of first and second molars. Overlap of the mesial surface of the mandibular third molar on the distal surface of the second molar. Buccal deflection of the third molar.

Fig. 13.—Lingual view. Buccal deflection.

The alignment of the buccal surface of the crown of the third molar with the crown of the second molar is determined by the interpretation of the occlusal surfaces, enamel caps, pulp chambers, and crown contacts of the second and third molars in the roentgenograph. Where there is no overlap of the third molar on the second molar, with the first and second molars showing normal contact, the third molar has no deflection. (Fig. 10.) An overlap of mesial surface

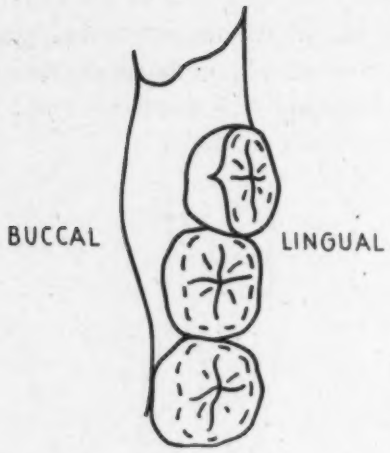


Fig. 14.

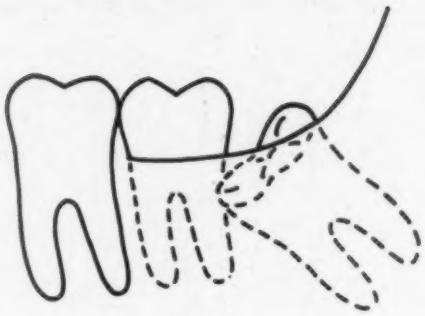
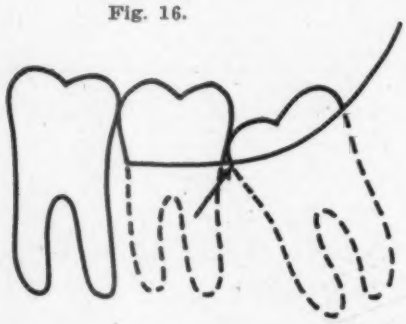


Fig. 15.

Fig. 14.—Occlusal view showing lingual deflection of the mandibular third molar.
Fig. 15.—Lingual view. First and second molars having normal contact. Occlusal surface of the mandibular third molar showing, indicating a lingual deflection.

Fig. 16.



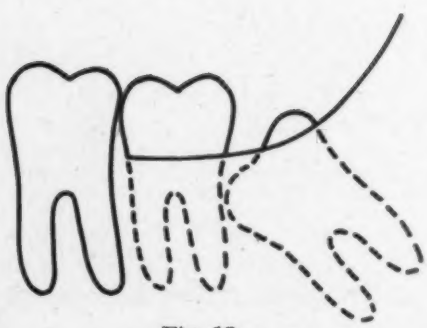


Fig. 17.

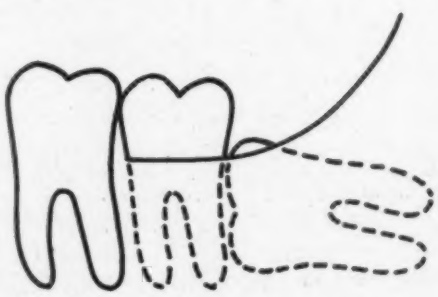


Fig. 18.

Fig. 16.—Mesial surface of the mandibular third molar accessible.
Fig. 17.—Mesial surface of the mandibular third molar inaccessible.
Fig. 18.—Mesial surface of the mandibular third molar inaccessible.

of the third molar upon the distal surface of the second molar, with the first and second molars showing normal contact points and clear enamel caps, indicates a buccal deflection of the third molar (Figs. 11, 12, and 13). A loss of the enamel cap of the third molar, showing a part of the occlusal surface of the third molar, with the first and second molars having a normal contact and clear enamel caps, indicates a lingual deflection (Figs. 14 and 15).

After an interpretation has been made of the position, size, and shape of the crown of the third molar, and also of its contact with the second molar, the character of the occlusal alignment of the third molar with the occlusal surface of the second is determined. The alignment of the occlusal surface of the third molar with the occlusal surface of the second molar is determined by drawing an imaginary line distally from the occlusal surface of the second molar. Variation in alignment from the imaginary line drawn distally from the occlusal surface of the second molar determines the amount of ossistructure covering the third molar. If the mesiobuccal surface of a vertical type of third molar is close to the line, the mesial surface is termed accessible to an exolever. If the mesial surface in any type is any appreciable distance below the line, it is spoken of as a mesial surface inaccessible to the application of an exolever.

The application of the exolever is usually made at the mesial, the mesiobuccal, or the buccal surface. When the exolever can be applied without any preliminary excision of the ossistructure, the case is then diagnosed as mesial surface accessible (Fig. 16). If excision of the ossistructure is necessary before an exolever can be applied, then the case is diagnosed as inaccessible (Figs. 17 and 18).

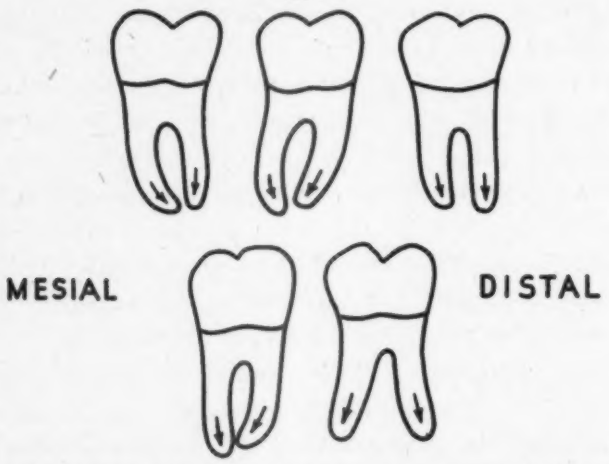
3. Character of the root formation

The roots of the mandibular third molar may vary in size from very short to exceedingly long; from being fused in a solid mass of a conical shape to two distinct roots markedly divergent, and the direction will vary from straight to a marked distal inclination. The most common root formations found with the impacted mandibular third molars are (Figs. 19 and 20):

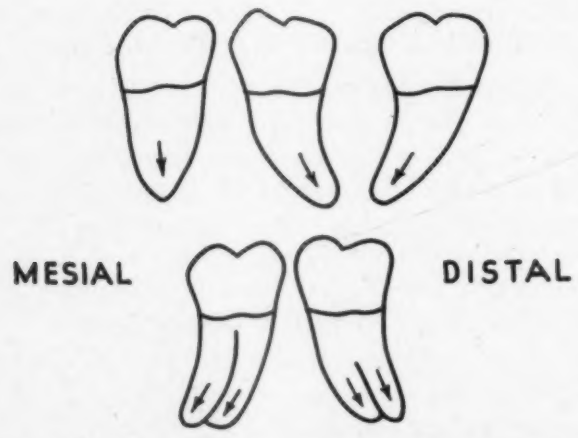
a. Both roots inclined distally.
b. Mesial root inclined distally and distal root straight.
c. Mesial root inclined distally and distal root inclined mesially.
d. Roots fused.
e. Both roots straight and separated.
f. Both roots fused and inclined mesially.
g. Both roots fused and inclined distally.
h. Mesial root straight and distal root inclined mesially.
i. Both roots inclined mesially.
j. Divergent roots.
k. Extra roots.
l. Hypercementosed roots.
m. Roots partially developed.

Contact of Roots.—In the vertical and the distoangular types of impacted mandibular third molars, there are cases where the mesial surface of the mesial root of the third molar is in contact with the distal or the distobuccal surface of the distal root of the second molar. Where there is such a contact, the pressure of the mesial root of the third molar during the exolever technique may fracture the distal root of the second molar, or, if the second molar is conical in shape, may displace the second molar.

Fig. 19.



ROOT FORMATIONS MANDIBULAR THIRD MOLARS



ROOT FORMATIONS MANDIBULAR THIRD MOLARS

Fig. 20.

4. INTERPRETATION OF THE OSSISTRUCTURE OF THE IMPACTED MANDIBULAR THIRD MOLAR

Where the mandibular third molar is not impacted and is in a normal position, the roentgenograph of the tooth and its supporting ossistructure will show the mesial, distal, lingual, and buccal surfaces to be free of ossistructure

to the gingival third of the tooth. The absence of ossistructure on these surfaces will allow the free application of the beaks of the forceps to the buccal and the lingual surfaces of the tooth. Thus the approximately normal position of the third molar shows no ossistructure beyond the gingival third of any surface of the tooth.

The ossistructure over the distoclusal surface of the third molar is quite a resisting factor in the removal of an impacted mandibular third molar.

In the vertical type of impaction, the ossistructure extends over the distoclusal surface of the third molar. The deeper the impaction, the more ossistructure covers the tooth.

Ramus.—The proximity of the ramus of the mandible to the impacted third molar indicates the amount of ossistructure covering the distoclusal surface of the third molar.

The roentgenograph outlines the cases where the ossistructure extends over the distoclusal surface.

The distance from the distal surface of the second molar to the ossistructure distal to the third molar determines the closeness of the ramus to the third molar.

The greater the distance of the ossistructure distal to the third molar to the distal surface of the second molar, the less will be the excision of the ossistructure distal to the third molar.

The ossistructure distal to the distoclusal surface of the vertical type of mandibular third molar, with the mesial surface inaccessible, almost completely covers the tooth.

The distoangular type is completely covered. As the vertical type emerges, more tooth is exposed and less ossistructure covers the tooth. The distoangular type becomes more and more embedded.

The ramus in some cases is on a level with the distoclusal surface of the second molar.

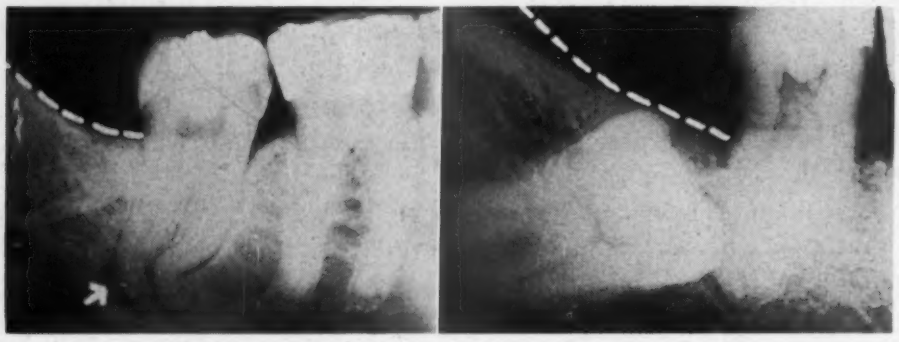
The other type of ramus is at a level below the distoclusal surface of the second molar and at a level with the gingival portion of the third molar.

Classification of Ramus Positions.—By classifying the extreme distal position of the ramus as Class I and the extreme mesial position as Class III, we find that Class II is the average position in which to find the anterior border of the ramus.

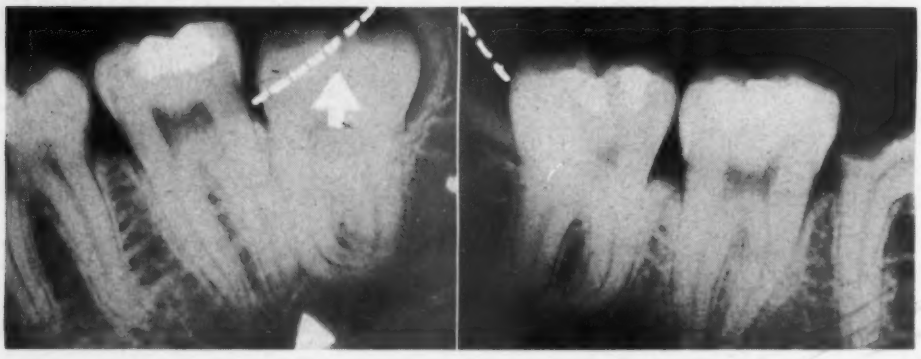
Class I ramus: The anterior border of the external oblique ridge of the mandible rises several millimeters distal to the crown of the normally erupted third molar. We find in this class that the third molar is fully erupted and the ossistructure distal to the crown continuing as a horizontal plane for some distance before rising to join the body of the ramus. There is no impingement of the mucosa distal to the tooth, and the tooth is free in all proportions. In this type of mandibular third molar, the ossistructure distal to the tooth is similar in every respect to a second molar after the removal of the third molar (Fig. 21).

With the mandibular third molar lying in an impacted position in Class I ramus, the ridge distal to the second molar would be well defined and present a

long section of normal gum tissue. The ossistructure in Class I ramus forming the buccal plate is thinner than the other types. The process distal to the crown of the third molar extends distally in a line parallel to the alveolar crest for some distance. (Fig. 22.)



Figs. 21 and 22.—Class I ramus.



Figs. 23 and 24.—Class II ramus.

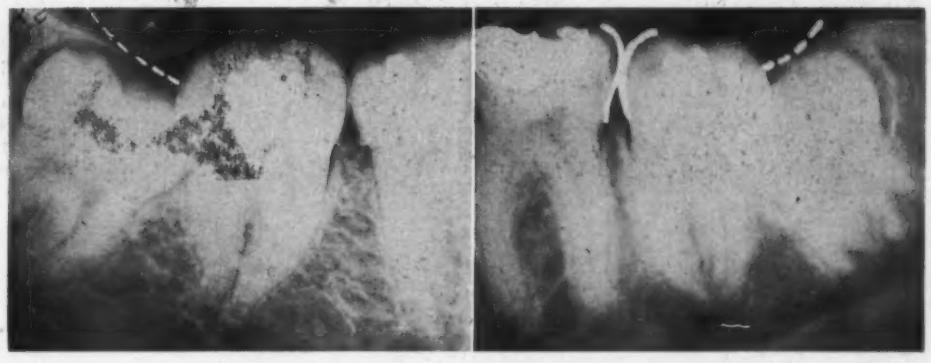
Class II ramus: The anterior border of the external oblique ridge rises from the distal surface of the second molar. The crown of the mandibular third molar, when in normal relation to the second molar, appears to be unerupted at the distoclusal surface. In the roentgenograph the ramus shadow is superimposed upon the distoclusal surface of the tooth. This may not be true, for the fibers of the external oblique ridge may be felt clinically merging with the body of the ramus just buccal to the second molar. Retraction of the cheek is thus limited by the close attachment of the mucosa to the buccal gingiva of the mandibular third molar. (Figs. 23 and 24.)

The ossistructure on the buccal surface is thick; the process distal to the third molar rises sharply and impinges on the distal surface of the third molar.

With the mandibular third molar impacted in this type of ramus, the buccal plate is quite thick and the impacted mandibular third molar lies partly in the body of the ramus.

Class III ramus: In this type the external oblique ridge rises from the groove of the second molar and at times at the mesial surface of the second

molar, even extending to the first molar. The third molar in this type is completely submerged. There is in this type a soft tissue involvement from trauma in an attempt to occlude with the upper third molar. The ramus rises abruptly from the distal surface of the second molar. The ossistructure in this type of ramus on the buccal surface may be said to be the ramus itself. The buccal plate is short and thick; this offers much resistance to the removal of the mandibular third molar. (Figs. 25 and 26.)



Figs. 25 and 26.—Class III ramus.

The Interseptum.—The interseptum between the distal root of the second molar and the mesial root of the third molar should be carefully studied. This is the ossistructure used as a fulcrum for the exolever. This structure should not be destroyed. It is a factor in support of the second molar after the third molar has been removed.

Mandibular Canal.—The mandibular canal when in proximity to the impacted mandibular third molar is in most instances outlined in the roentgenograph.

As the alveolar nerve comes in contact with the third molar, both may be changed—the nerve in the line of progress and the tooth in form. Why, in the development of tooth substance, there is an alteration of form due to the presence of the nerve is difficult to explain, but, in many cases, the entire shape of the root or roots conforms in a marked degree to the presence of the nerve. This occurs in the distal curvature of the roots of a horizontal impaction, where the roots in many cases take the form of the canal on one of their lateral surfaces, resulting in a definite notch or shoulder in the side of the root. In rare instances, the two roots of a vertical type of impaction encircle the canal and become fused at their apices, thus forming a complete circle of tooth substance around the nerve.

If the root apices can be clearly seen in the roentgenograph, then the roots are not in the canal; if the root apices are hazy and not clearly defined in the roentgenograph, then they are in the canal.

Since reflex disturbances may be of dental origin, the impingement of the roots of the third molar upon the contents of the inferior dental canal cannot be overlooked.

DESCRIPTION OF TYPES OF MANDIBULAR THIRD MOLARS



Fig. 27.



Fig. 28.

Fig. 27.—*Vertical type,* first example. No deflection. Mesial surface accessible. Both roots inclined distally. Superior border of the ossistructure at the gingival third of the third molar. Ramus type one. Mandibular canal lies beneath the apices of the roots of the third molar. Second molar in normal position. Caries in first molar.

Fig. 28.—*Vertical type,* second example. No deflection. Mesial surface accessible. Both roots fused, with sharp distal inclination at right angle to the body of the tooth. Superior border of the ossistructure at the distoclusal surface of the third molar. Ramus type two. Mandibular canal lies beneath the curvature of the root. Caries in the distal surface of the third molar. Second molar carious, roots fused and conical in shape. The application of an exolever at the mesial surface of the third molar using the second molar as a fulcrum will force the second molar out of its socket before the third molar is elevated. The ossistructure distal to the third molar will prevent the dislodgment of the third molar.



Fig. 29.



Fig. 30.

Fig. 29.—*Vertical type,* third example. No deflection. Mesial surface accessible. Mesial root inclined distally, distal root straight. Superior border of the ossistructure covering all of the crown of the third molar with the exception of the mesioclusal surface. Ramus type two. Mandibular canal lies beneath the roots of the third molar. Second molar normal. First molar missing.

Fig. 30.—*Vertical type,* fourth example. No deflection. Mesial surface accessible. Both roots inclined mesially. Superior border of the ossistructure at the gingival third of the third molar. Ramus type one. Mandibular canal lies beneath the roots of the third molar. Second molar normal.

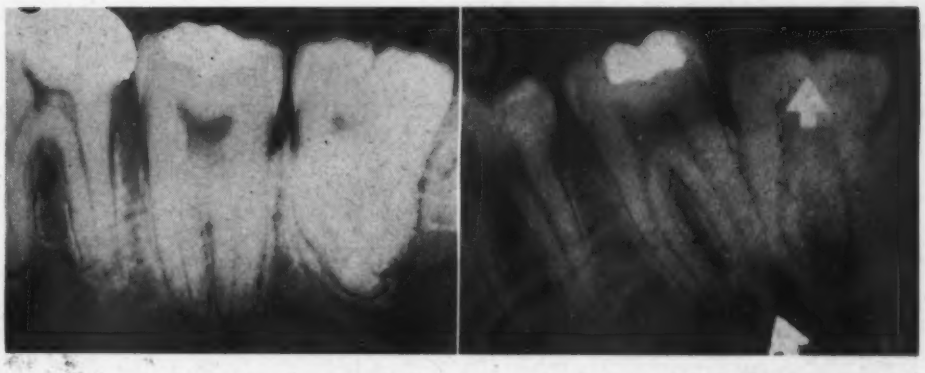


Fig. 31. Fig. 32.

Fig. 31.—*Vertical type,* fifth example. No deflection. Mesial surface accessible. Roots fused with the bifurcation of the roots in the inferior third of the root. The apex of the root inclined distally and not in the mandibular canal. The superior border of the ossistructure at the distoclusal surface of the third molar. Ramus type two. Second molar normal. The lingual plate of the ossistructure is mechanically locked in the low bifurcation of the root of the third molar. This locking of the roots around the ossistructure acts as a hindrance to the removal of this tooth.

Fig. 32.—*Vertical type,* sixth example. No deflection. Mesial surface accessible. Mesial root inclined distally, distal root straight and fused to the distal curvature of the mesial root. Contact of the mesial root of the third molar with the distal surface of the second molar. Bifurcation of the root near the inferior, or apical portion of the root. Superior border of the ossistructure covering most of the distoclusal surface of the third molar. Ramus type two. Mandibular canal lies beneath the roots of the third molar. The displacement of this type of tooth distally in its socket results in pressure of the mesial root of the third molar upon the distal root of the second molar with possible fracture of the second molar. The ossistructure distal to the third molar offers enough resistance to effect a possible fracture of the mandible if the third molar is forced distally before the removal of the overlying ossistructure.

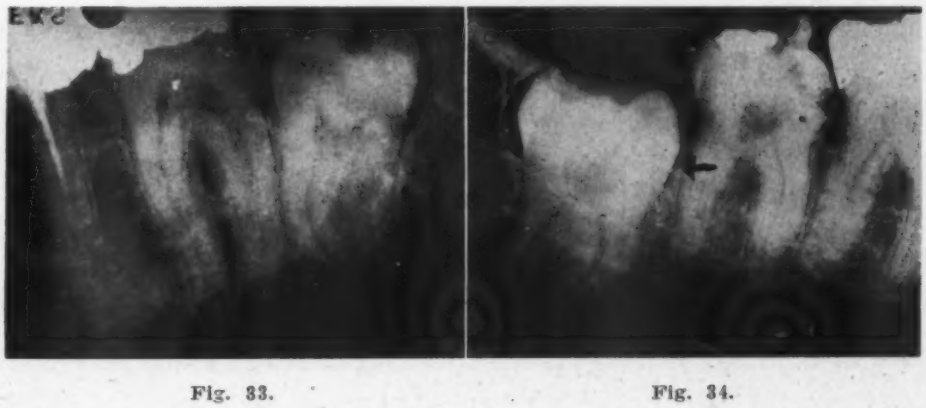


Fig. 33. Fig. 34.

Fig. 33.—*Vertical type,* seventh example. No deflection. Mesial surface accessible. Mesial root inclined distally, distal root straight. Contact of the mesial root of the third molar with the distal root of the second molar. Superior border of the ossistructure almost completely covering the crown of the third molar with the exception of the extreme corner of mesioclusal surface. Ramus type two. Root apices show clearly. The root apices are either to the buccal or the lingual of the mandibular canal.

Fig. 34.—*Vertical type,* eighth example. No deflection. Mesial surface inaccessible. Mesial root inclined distally, distal root straight. Ossistructure completely covers the crown of the third molar. Ramus type two. Interseptum between the third molar and the second molar clearly defined. Mandibular canal passes the tooth midway between the gingiva and the apex. The canal shows clearly and passes the tooth either to the buccal or the lingual. Second molar in normal position. Distal root of second molar shows clearly, probably not in the canal. Mesial root of second molar hazy and not clearly defined, probably in the canal.

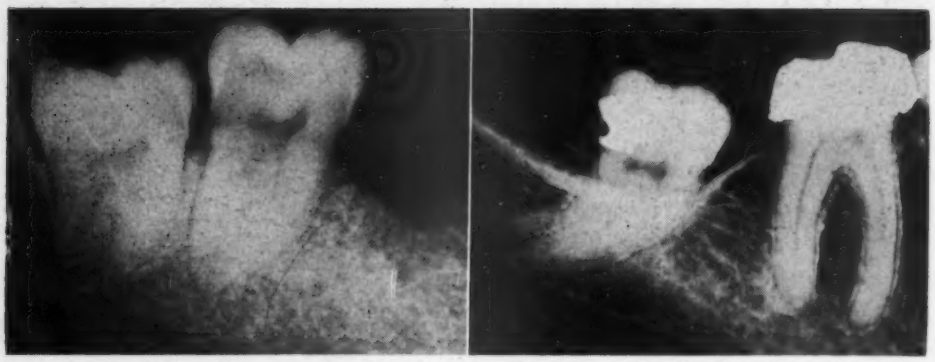


Fig. 35. Fig. 36.

Fig. 35.—*Vertical type,* ninth example. No deflection. Mesial surface inaccessible. Mesial root straight, apex clear. Distal root hazy and probably absorbed by pressure upon the canal. Ossistructure almost completely covers crown of the third molar. Ramus type two. Interseptum between the third molar and the second molar definitely defined. Second molar conical in shape. First molar missing.

Fig. 36.—*Vertical type,* tenth example. Isolated third molar. Drifting of the third molar where the second molar has been extracted. Both roots inclined distally. The crown of the third molar exposed on all surfaces. Ramus type one. Mandibular canal beneath the mesial surface of the root of the third molar. First molar in normal position.

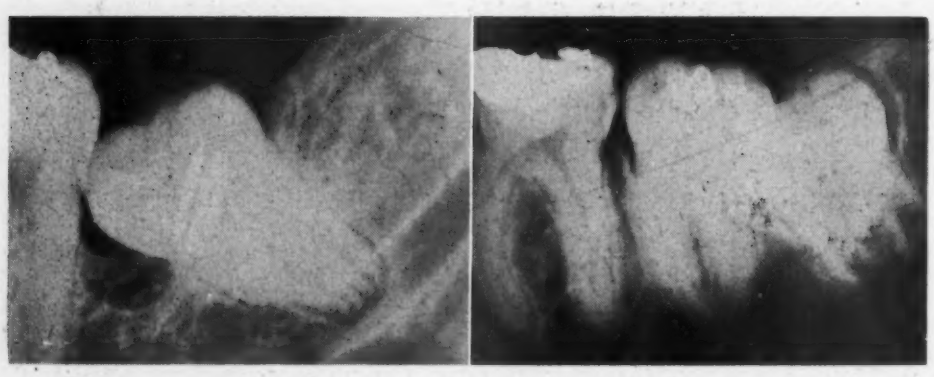


Fig. 37. Fig. 38.

Fig. 37.—*Mesioangular type,* first example. No deflection. Mesial surface accessible. Both roots straight, apices hazy and probably in mandibular canal. Distal surface of the crown of the third molar free of ossistructure to the gingival third of the tooth. Pathology present at the mesial surface of the third molar. Contact of the mesioclusal surface of the third molar with the distal surface of the second molar.

Fig. 38.—*Mesioangular type,* second example. Buccal deflection. Roots partially developed. Ossistructure completely covers crown of the third molar. Mandibular canal lies beneath undeveloped roots of the third molar. Second molar normal.

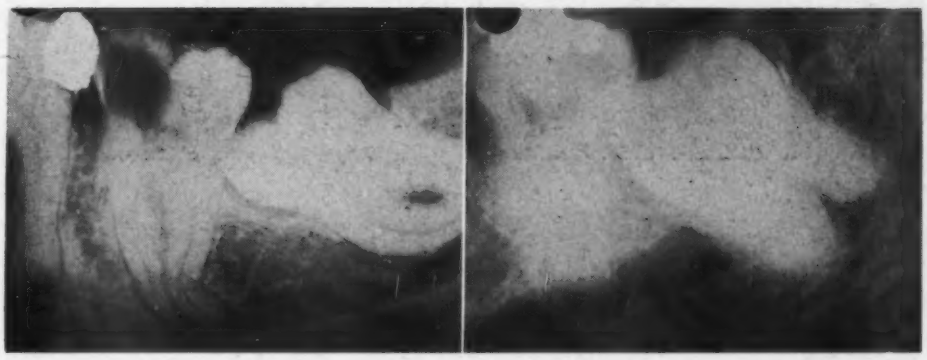


Fig. 39. Fig. 40.

Fig. 39.—*Mesioangular type,* third example. No deflection. Mesial root inclined distally, distal root mesially. The lingual plate is locked mechanically in the bifurcation at the lower third of the roots of the third molar. Superior border of the ossistructure at the distogingival portion of the third molar. Ramus type one. Contact of mesioclusal surface of the third molar with the distal surface of the second molar. Decay in mesial surface of second molar. Roots of second molar inclined distally. The distal root is hazy and probably in the canal.

Fig. 40.—*Mesioangular* type, fourth example. Lingual deflection (occlusal surface of the crown of the third molar showing). Mesial surface inaccessible. Roots straight. Ossistructure completely covers the third molar. Superior border of the ossistructure starts at the distal surface of the second molar. Ramus type three. Roots of the second molar fused. Roots show clearly and pass either to the lingual or the buccal of the mandibular canal.

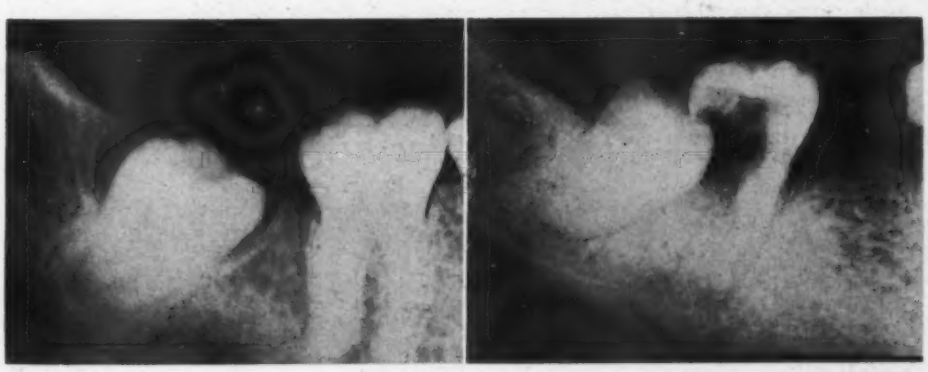


Fig. 41. Fig. 42.

Fig. 41.—*Mesioangular type,* fifth example. Isolated. Mesial surface inaccessible. Roots partially developed. Ossistructure covering the third molar. Mesial drifting of third molar, a result of the removal of the second molar.

Fig. 42.—*Mesioangular type,* sixth example. Drifting of the third molar into the distal surface of the second molar. Distal root straight, mesial root inclined distally. Third molar free of ossistructure on the distal surface. Decay of second molar. Distal root of the second molar entirely separate from the second molar.





Fig. 43. Fig. 44.

Fig. 43.—*Mesioangular type,* seventh example. Buccal deflection. Mesial surface inaccessible. Third molar completely covered by ossistructure. Pressure of the mesioclusal surface of the third molar against the apex of the root of the second molar.

Fig. 44.—*Mesioangular type,* eighth example. Isolated. Mesial root inclined distally, distal root straight. Third molar completely covered by ossistructure. Mandibular canal close to the root apices.



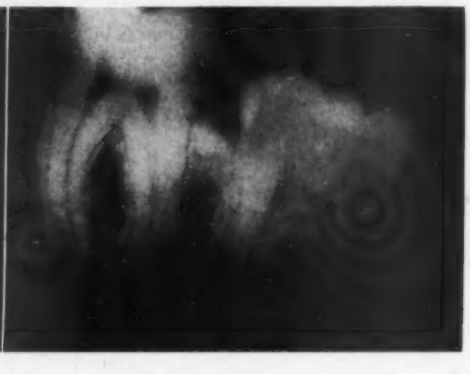


Fig. 45. Fig. 46.

Fig. 45.—*Distoangular type,* first example. Lingual deflection. Mesial surface accessible. Roots fused, conical in shape, and in contact with the distal root of the second molar. Superior border of the ossistructure covers the crown with the exception of the mesioclusal surface. Ramus type two. Rarefaction of the ossistructure distal to the distoclusal surface of the third molar. Root apex showing clearly and not in the mandibular canal. Second molar nonvital.

Fig. 46.—*Distoangular type,* second example. Mesial surface inaccessible. Apices of roots fused, with the bifurcation at the inferior third of the roots. Interseptum between the distal surface of the second molar and the mesial surface of the third molar rather more than usual. Crown of the third molar completely embedded in the ascending ramus.





Fig. 47. Fig. 48.

Fig. 47.—*Distoangular type,* third example. Isolated. This type is usually missed in the ordinary lingual view roentgenograph. The third molar is in the ascending ramus. Distal root inclined mesially and showing clearly and not in the mandibular canal. Mesial root straight and apex hazy, probably in the canal. Distal root passes either to the lingual or to the buccal side of the mandibular canal.

Fig. 48.—*Distoangular type,* fourth example. Isolated. Roots straight. Ossistructure completely covers the third molar. Mandibular canal beneath the roots of the third molar.





Fig. 49. Fig. 50.

Fig. 49.—*Horizontal type,* first example. No deflection. Mesial surface accessible. Roots fused. Superior border of the ossistructure covers most of the third molar with the exception of the distoclusal surface. Ramus type two. Interseptum separates the third molar from the second molar. Mandibular canal lies beneath the mesial surface of the third molar. No contact of the occlusal surface of the third molar with the distal surface of the second molar. Second molar normal.

Fig. 50.—*Horizontal type,* second example. No deflection. Mesial surface inaccessible. Roots fused. Superior border of the ossistructure at the gingival third of the distal portion of the third molar. Ramus type one. Second molar normal. Mesioclusal surface of the third molar in contact with the distal root of the second molar.



Fig. 51.—*Horizontal type,* third example. Mesioclusal surface deflected buccally. Mesial surface inaccessible. Roots completely fused. Superior border of the ossistructure begins at the middle of the second molar and completely covers the third molar. Ramus type three.





Fig. 52. Fig. 53.

Fig. 52.—Complete lingual impaction of the mandibular third molar. Occlusal surface showing.

Fig. 53.—Postoperative film of Fig. 52, showing sockets in the buccal plate. The tooth was lying in a transverse-horizontal position—the occlusal surface to the lingual and the roots to the buccal plate.

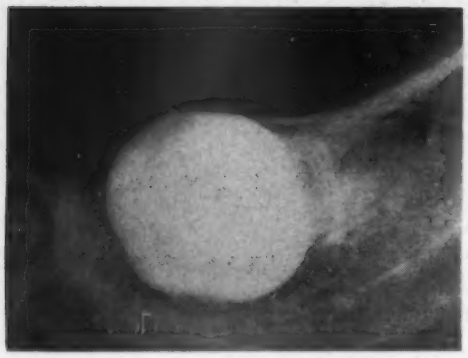


Fig. 54.—Isolated lingual impaction of the mandibular third molar.



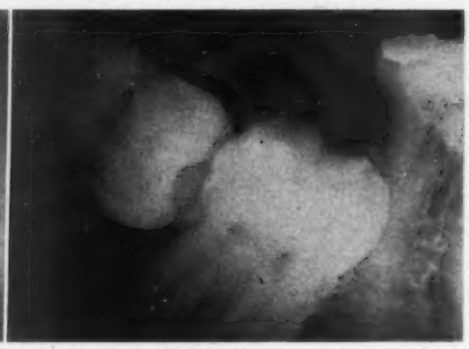


Fig. 55. Fig. 56.

Fig. 55.—Horizontal impaction of the second and third molars.
Fig. 56.—Mesioangular impaction of the second molar with a horizontal impaction of the third molar.

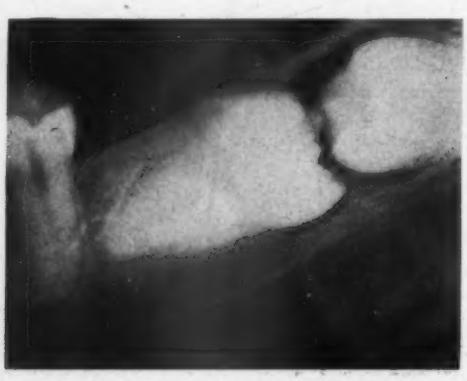


Fig. 57.—Horizontal impaction of the second molar with the occlusal surface inclined distally and the roots mesially. Horizontal impaction of the third molar with the occlusal surface inclined mesially and the roots distally. Contact of the occlusal surfaces of both the second and third molars.

Part V. The Removal of the Erupted Mandibular Third Molar

The removal of a fully erupted mandibular third molar does not as a rule present many difficulties—unless the tooth has marked inclinations of the roots or hypercementosed roots.

The exolever technique used is based upon the inclination of the roots. The instruments used are the Winter exolevers or displacing instruments, Nos. 1, 2, 3, and 4, which are applied at the mesiobuccal surface of the mandibular third molar. The instruments are numbered according to size, No. 1 being the smallest. After enough space has been made with the No. 1, a No. 2, 3, or 4 should be used as required.



Fig. 58.



Fig. 59.

Fig. 58.—Application of the exolever at the mesial surface of the mandibular third molar.
Fig. 59.—Application of the exolever at the buccal surface in the bifurcation of the third molar.

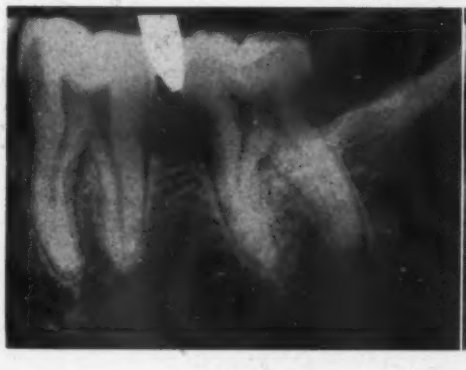


Fig. 60.



Fig. 61.

Fig. 60.—Incorrect application of the exolever, using the second molar as a fulcrum.
Fig. 61.—Incorrect application of the exolever, destroying the ossistructure distal to the second molar.

Because the tooth is not supported on the distal surface by another tooth, the application of the Number 1 L. or Number 1 R. Winter exolever at the mesiobuccal surface, using the ossistructure distal to the second molar as a fulcrum, will elevate the tooth. The L. and R. signify left or right. The point of the exolever is wedged between the mesial surface of the third molar and the

ossistructure distal to the second molar, and forced downward (Fig. 58). A slight rotation of the handle of the exolever to the mesial loosens the third molar. The second molar is not used as a fulcrum. The conical-shaped mandibular third molar and the mandibular third molar with the roots deflected distally are easily removed by this method.



Fig. 62.



Fig. 63.

Fig. 62.—Exolever technique. The application of the exolever at the mesial surface of the mandibular third molar. The tooth is elevated distally.

Fig. 63.—After the tooth is elevated distally to overcome the resistance of the root curvature, an exolever is applied at the buccal surface opposite the bifurcation. A buccal application elevates the tooth upward and out of the socket.



Fig. 64.



Fig. 65.

Fig. 64.—Tooth removed. Debris at the mesial surface of the socket. These particles are the cause of postoperative discomfort.

Fig. 65.—Clean socket.

In cases where the distal root offers resistance, the exolever at the mesiobuccal surface is used to overcome the resistance of the root curvature. After the third molar has been directed distally and upward to overcome the curvature of the mesial root, the Winter exolever No. 10 L. or 10 R. is used for a buccal application (Fig. 59). This exolever is applied at the bifurcation of the roots. The ossistructure on the buccal surface is used as a fulcrum. To gain access to the bifurcation the Brown exolever with cutting blades is forced through the ossistructure at an angle from the superior border to the bifurcation. The Winter exolever No. 10 or 12 is then placed into the bifurcation. A secure fulcrum is obtained, a downward pressure is exerted, pushing the point into the

bifurcation. The instrument acts as a wedge between the buccal surface of the ossistructure to such an extent that the tooth is elevated out of the socket.

If resistance is encountered, the pressure is increased and the handle of the exolever is turned slightly buccally and downward. The back of the blade will rest on the ossistructure at the buccal surface. Repeated movements at this point will prevent a fracture of the distal root of the third molar.

If, however, the distal root of the third molar is fractured, it is easily removed by applying a No. 12 or 14 Winter exolever in the mesial socket, engaging the point of the exolever at the mesial surface of the distal root. A slight turn of the handle mesially dislodges the root. The exolever acts as an ossisector when passing from the mesial socket to the distal root, dissecting the ossistructure between the sockets.

The exolever technique described for the fully erupted mandibular third molar is the same as is used for the impacted mandibular third molar. The mesial application is used to overcome the resistance of the inclination of the roots and the buccal application to remove the tooth in a vertical position.

The exolever is not wedged between the second and third molars. The second molar is not used as a fulcrum (Fig. 60).

The ossistructure distal to the second molar is not destroyed (Fig. 61). The wedging of the exolever is accomplished in the space between the ossistructure distal to the second molar and the mesial surface of the third molar.

Part VI. The Removal of the Impacted Mandibular Third Molar

The removal of an impacted mandibular third molar should be regarded in every sense as a surgical procedure. The removal is accomplished in either of two ways:

1. Removing the ossistructure distal to the third molar, creating a space distal to the third molar to permit the elevation of the tooth into that space in the line of least resistance (Figs. 66 and 67).

2. Sectioning the crown and the roots and removing the tooth in sections (Figs. 68 and 69).

In many cases a combination of both methods may be employed. It does not matter whether the tooth is removed in toto or in sections. The ideal operative procedure is to remove the mandibular third molar without the destruction of too much ossistructure and without harming the neighboring teeth.

The chief aim in the removal of the ossistructure over the crown of the tooth and the excision of the ossistructure distal to the third molar is to permit the elevation of the tooth in the line of least resistance.

1. Distal Ossistructure Excision Technique

a. Gum tissue (mucoperiosteal flap) technique.
b. Retraction of the mucoperiosteum.
c. Examination of the ossistructure surrounding the crown.
d. Removal of the ossistructure over the crown.
e. Excision of the ossistructure distal to the crown.

f. Excision of the ossistructure mesial to the crown to obtain access for the exolever at the mesiobuccal surface.

g. Excision of the ossistructure on the buccal surface to obtain access for the buccal application of the exolever.

h. Application of the exolever at the mesiobuccal surface and the elevation of the tooth into the space created distal to the third molar.

i. Application of the exolever at the buccal surface for the final delivery of the tooth.

j. Cleansing of socket of all debris.

k. Suturing of the mucoperiosteal flap.



Fig. 66.

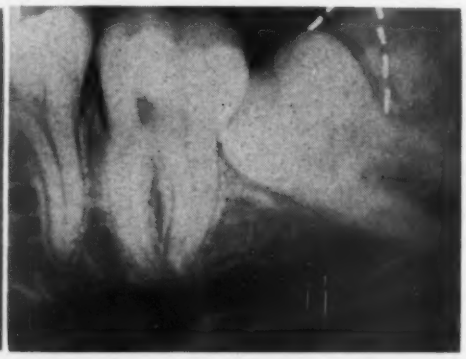


Fig. 67.

Fig. 66.—Distal excision technique. Showing the amount of ossistructure to be removed to permit the elevation of the third molar into the space created distal to it.

Fig. 67.—Distal excision technique. Showing the amount of ossistructure to be removed to permit the elevation of the third molar into the space created distal to it.

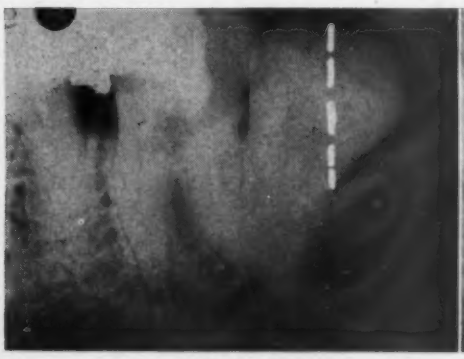


Fig. 68.



Fig. 69.

Fig. 68.—Sectioning technique. Showing portions of the mandibular third molar to be split to create space for elevating the tooth.

Fig. 69.—Sectioning technique. Showing portions of the mandibular third molar to be split to create space for elevating the tooth.

a. *Gum Tissue Technique.*—An impacted mandibular third molar should always be removed from the buccal and not from the lingual approach. The lingual tissue must be regarded with care and left undisturbed. The muco-

periosteal flap is designed to expose the operative area and not to interfere with the lingual tissue.

Examination of the mandible shows the internal oblique line of the mandible to be deflected buccally. If an incision is started at the distolingual surface of the second molar and extended in a direct line, it will enter the soft tissue of the throat.



Fig. 70.—Incision for flap. Distal incision extends from the distolingual surface of the second molar distally toward the buccal.



Fig. 71.—Incision. The buccal incision at the mesial surface of the second molar.

The incision to gain maximum access to the ossistructure starts at the distolingual surface of the second molar and extends distally to the buccal surface, following the line of the ridge between the internal and external oblique ridges (Fig. 70). The buccal incision is made at the mesiobuccal surface of the second molar, extending from the gingiva into the mucobuccal fold (Fig. 71).

b. *Retraction of the Mucoperiosteal Flap.*—The flap is carefully dissected from around the distal surface of the second molar. The gingival tissue on the distal and the buccal surfaces of the second molar is included in the flap. The mucoperiosteum is retracted with a periosteal elevator from the ossistructure. Traumatization should be avoided as this tissue flap when replaced forms the protection for the ossistructure, the second molar, and the socket after the removal of the tooth.

c. *Examination of the Ossistructure Surrounding the Crown.*—After retraction of the mucoperiosteal flap, we find that the tooth may or may not be covered with ossistructure. It is impossible to state preoperatively from a roentgenograph whether or not the tooth is completely covered with ossistructure.

d. *Removal of the Ossistructure Over the Crown.*—Any ossistructure over the crown is now removed by means of the ossisector, hand chisel, bone drills, or impactor, exposing completely the occlusal surface of the tooth.

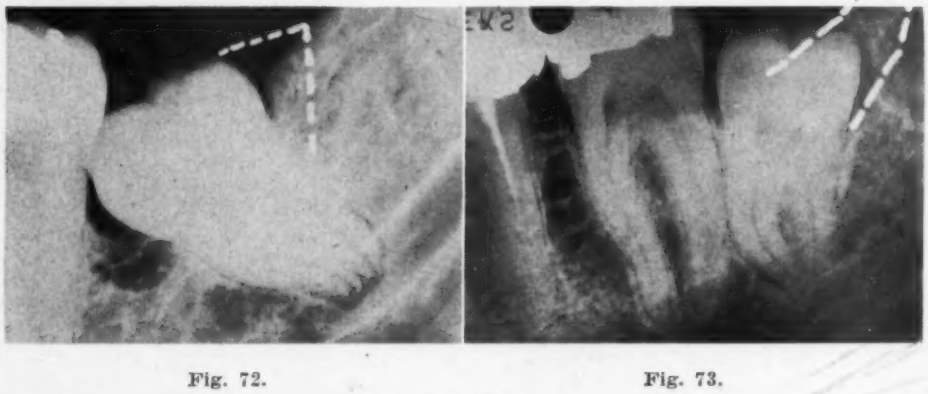


Fig. 72. Fig. 73.

Fig. 72.—The ossistructure from the distoclusal surface of the third molar is excised to the gingival third of the tooth. The space thus created distal to the third molar allows elevation of the tooth.

Fig. 73.—The ossistructure from the distoclusal surface of the third molar is excised to the gingival third of the tooth. The space thus created distal to the third molar allows elevation of the tooth.

e. *Excision of the Ossistructure Distal to the Crown.*—The inclination of the root formation determines the amount of distal excision of the ossistructure. The ossistructure extending from the occlusal surface to the gingival third is excised to allow the tooth to be elevated into a vertical position (Fig. 72 and 73). The purpose of the distal excision is to create a space to permit the tooth to be directed distally far enough to overcome the resistance of root curvature.

No excision of the distal ossistructure is indicated where the distal surface of the tooth is free of ossistructure from the occlusal surface to the gingival third of the tooth.

f. *Excision of the Ossistructure Mesial to the Crown to Obtain Access for the Exolever at the Mesiobuccal Surface.*—The mesiobuccal excision of the ossistructure is indicated on the mesiobuccal aspect to allow the application and execution of the exolever movement to the mesial surface of the third molar.

The ossistructure is cut at an angle from the superior border of the ossistructure to the tooth. The superior border is not excised, as this acts as a fulcrum for the exolever.

No excision of the ossistructure is made below the mesial surface of the third molar crown. An excision below the mesial surface of the crown decreases the value of the superior border of the ossistructure as a fulcrum at this point.

g. *Excision of the Ossistructure on the Buccal Surface to Obtain Access for the Buccal Application of the Exolever.*—The extent to which the tooth can be displaced distally and upward will determine whether or not a buccal excision of ossistructure is necessary. Where the tooth cannot be elevated and removed by a mesial application of the exolever, an excision is made on the buccal surface opposite the bifurcation. This excision is made after the elevation of the tooth distally. This permits access of the exolever for final delivery of the tooth.

The excision is made from the buccal aspect to the bifurcation of the roots and at such an angle as to preserve the superior border of the ossistructure as a fulcrum. It is very important that the excision be no lower than the bifurcation. If it is below the bifurcation, the fulcrum is lost.

At times the buccal excision may be continuous with the distal excision.

h. *Application of the Exolever at the Mesiobuccal Surface and Elevation of the Tooth Into the Space Created Distal to the Third Molar.*—After the ossistructure on the occlusal, distal, mesiobuccal, and buccal surfaces has been excised, the exolever is applied to the mesial surface of the third molar, using the ossistructure distal to the second molar as a fulcrum.

Exolevers are the Winter type Nos. 1, 2, 3, or 4.

The blade is first applied to the mesiobuccal surface; the handle is then directed buccally and the blade adjusted to the mesial surface.

When the adjustment has been made, the handle of the exolever is turned mesially and the tooth partially raised distally and upward out of the socket if the root inclination will permit. As the space increases and the fulcrum is lost, a larger exolever blade is applied. In elevating the tooth, do not disturb the distal surface of the second molar.

i. *Application of the Exolever at the Buccal Surface for the Final Delivery of the Tooth.*—When the root formation indicates that the tooth is not to be elevated completely out of the socket by the mesial application of the exolever alone, a buccal application is made with a Winter exolever No. 10 to complete the removal of the tooth. The point of the blade is directed into the buccal surface toward the bifurcation of the roots and is sent down on this surface to act as a wedge between the ossistructure and the tooth. The wedging should elevate the tooth. If this fails, the blade is wedged further and the handle is turned buccally to complete the removal of the tooth.

All restricting ossistructure must be removed for complete elevation of the tooth. If the tooth does not elevate easily, the areas of osseous obstruction should be examined before too much force is used.

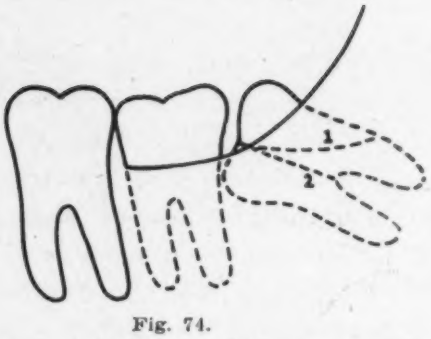


Fig. 74.

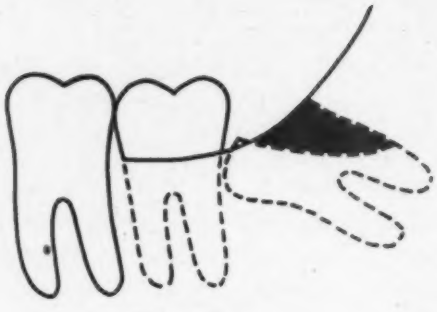


Fig. 75.

Fig. 74.—Tooth sectioning. Tooth may split: (1) distal portion of crown; (2) complete mesial root.

Fig. 75.—Removal of distal section.

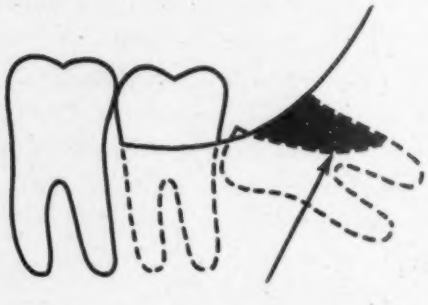


Fig. 76.

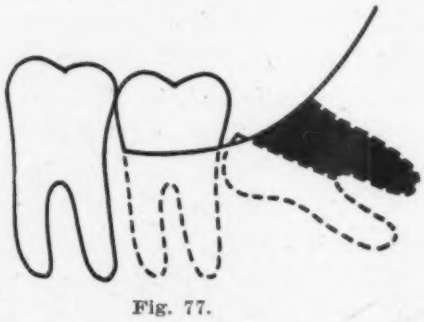


Fig. 77.

Fig. 76.—Removal of remaining distal root by inserting a Winter exolever No. 12 into pulp chamber.

Fig. 77.—Distal root removed.

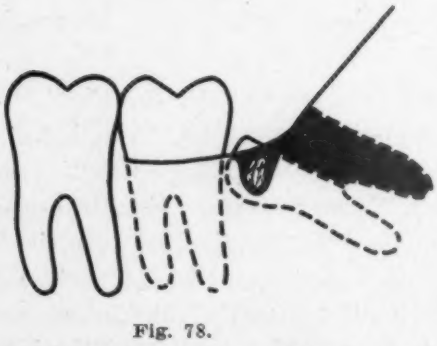


Fig. 78.

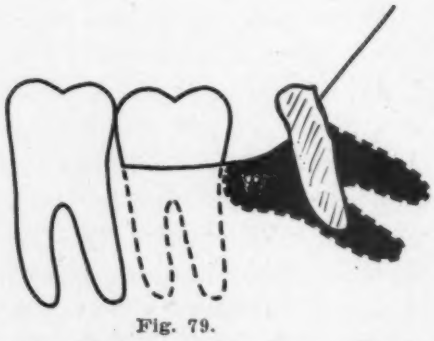


Fig. 79.

Fig. 78.—Channel at an angle from the superior border of the ossistructure to the mesial surface of the mandibular third molar.

Fig. 79.—Final delivery of the mesial root.

j. *Cleansing of the Socket.*—All particles of tooth, ossistructure, and debris are removed from the socket. The capsule, if present, should be removed. The edges are smoothed with files. A flow of blood is stimulated into the cavity.

k. *Suturing the Flap.*—The attached mucoperiosteum on the mesial surface of the table of ossistructure is slightly elevated to facilitate the insertion of the needle for final suturing. If this is not done, the needle will tear the tissue on this surface. Sutures are tied lightly.

2. SECTIONING OF THE CROWN AND ROOTS AND REMOVING THE TOOTH IN SECTIONS

The gum tissue technique, retraction of the mucoperiosteum, and examination and removal of the ossistructure over the crown of the tooth are the same in the sectioning, or tooth division technique as in the excision of the distal ossistructure technique.

Realizing that the retentive factor in the removal of an impacted tooth is the ossistructure, it remains either to take away enough of the retentive structure and extract the tooth in one piece or to divide the tooth and remove it in sections. The less ossistructure removed, the less pain will follow the removal. In many cases the tooth can be removed with very little excision of the ossistructure.

Tooth division is accomplished after the ossistructure over the crown is removed. No attempt should be made to elevate the tooth before sectioning. A loose tooth will not section. The use of the impactor or the automatic chisel has made the sectioning of teeth very easy.

The splitting chisel is placed at the buccal notch, parallel to the long axis of the tooth; pressure is applied to the chisel, and with one stroke the tooth is sectioned. The tooth may fracture at one of two places. Either the whole mesial root is split, or the distoclusal portion of the tooth is split. (Fig. 74.)

If the whole distal section is divided, it is removed with a Winter exolever No. 1 (Fig. 75).

Should the distal portion be fractured at the neck, the crown portion is removed. The distal root is removed with a Winter exolever No. 12. This is accomplished by placing the point of the exolever in the pulp chamber and applying pressure toward the mesial side (Figs. 76 and 77).

The excision of the ossistructure at the mesiobuccal surface is made as in the previous technique. The excision is made at an angle, so that the superior border acts as a fulcrum for the back of the exolever shank. The channel at the mesiobuccal surface is made with a chisel or the Brown ossisector (Fig. 78).

The final delivery of the mesial root is made with the Winter exolevers Nos. 1, 2, 3, or 4. The mesial portion of the tooth is elevated into the space created by the portion of the tooth sectioned away. Most of this operative technique is performed on the tooth itself (Fig. 79).

After the particles of tooth have been removed, the capsule is dissected out. All fragments are removed; sharp edges are smoothed with files. The flap is then sutured as described in the previous technique.

Case 1.—Mesioangular Type of Mandibular Third Molar. Sectioning Technique.—



Fig. 80.



Fig. 81.

Fig. 80.—Mesioangular type of mandibular third molar, no deflection, mesial surface inaccessible, mesial root inclined distally, distal root straight.

Fig. 81.—Sectioning of the distoclusal surface of the mandibular third molar.



Fig. 82.



Fig. 83.

Fig. 82.—Removal of the distoclusal surface of the mandibular third molar, creating a space distal to the third molar for the elevation of the tooth into the space created.

Fig. 83.—Elevation of the mandibular third molar into the space created by sectioning of the distal surface of the third molar. Tooth removed by a buccal application at the bifurcation of the tooth.

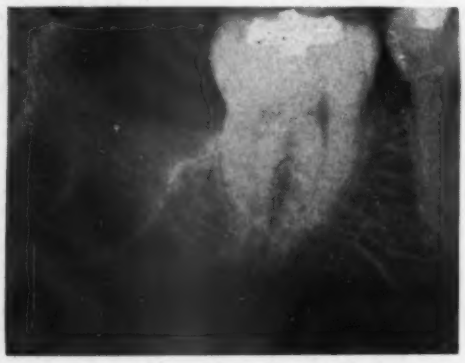


Fig. 84.—Socket after tooth removal.

Case 2.—Mesioangular Type of Mandibular Third Molar. Sectioning Technique.—

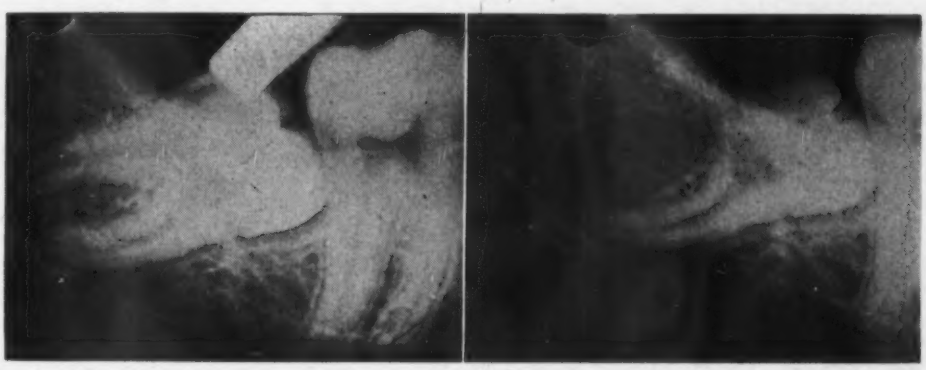


Fig. 85. Fig. 86.

Fig. 85.—Mesioangular type of mandibular third molar, no deflection, mesial surface inaccessible, mesial root inclined distally, distal root inclined mesially. Splitting chisel in position for sectioning of tooth.

Fig. 86.—Distal root removed.

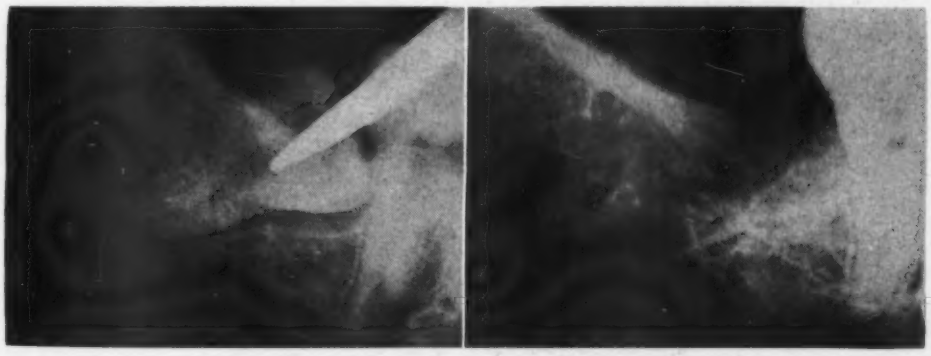


Fig. 87. Fig. 88.

Fig. 87.—Exolever in channel from superior surface of the ossistructure to the mesial surface of the mandibular third molar.

Fig. 88.—Socket showing tooth removed. Very little ossistructure removed.

Case 3.—Horizontal Type of Mandibular Third Molar. Sectioning Technique.—

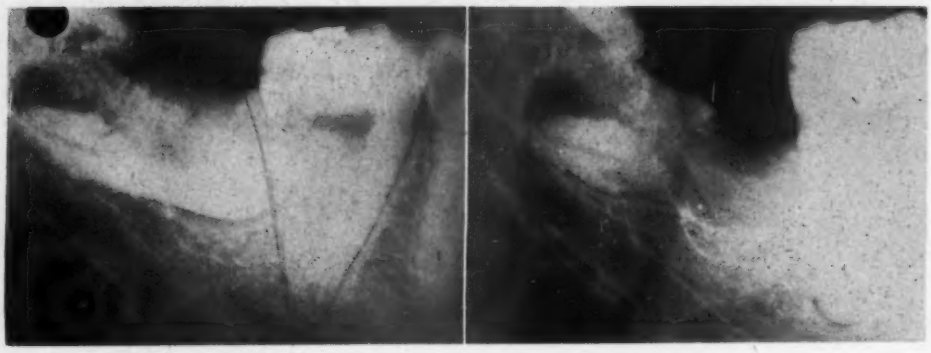


Fig. 89. Fig. 90.

Fig. 89.—Distal root removed.
Fig. 90.—Removal of crown. Mesial root still in place.



Fig. 91.—Mesial root elevated forward into space created by the removal of the mesial portion of the crown, and root removed.

Case 4.—Horizontal Type of Mandibular Third Molar. Sectioning Technique.—



Fig. 92.—Stages in the technique of sectioning the mandibular third molar.

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EFFECT OF PERIAPICAL PROCESSES OF DECIDUOUS TEETH ON THE BUDS OF PERMANENT TEETH

Pathological-Clinical Study

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THE purpose of this pathological-clinical study is to explain the etiology of defects of the enamel and other structures observed in individual permanent teeth not associated with any systemic disturbance of the calcium-phosphorus metabolism or a mechanical traumatization during the development. This investigation also offered the opportunity to observe the difference between chronic periapical infections of deciduous teeth and those of the permanent teeth. The conclusions drawn from these findings will be strengthened by the report on a pertinent clinical case.

Turner[1] described defects of the enamel of two permanent premolars and traced them to an apical infection of the nearest deciduous molar. The attempts to prove microscopically the pathogenesis of enamel "hypoplasias" of individual teeth failed because of the lack of adequate autopsy material and the inadequacy of approaching this problem by experimental work.

It was not until 1932 that Bauer[2] described the changes which extended from the chronic periapical process of two deciduous mandibular molars to the united epithelium of the buds of the permanent premolars. The specimen was removed at autopsy from a 5½-year-old girl who died of pneumonia. The roentgenographic examination of the mandible showed extensive periapical and interradicular processes of the badly decayed deciduous second molars, which extended down to the underlying buds.

The microscopic study of these parts of the mandible revealed a gangrene of the pulps of the deciduous molars. This gangrene resulted in a rather localized osteomyelitis which enveloped the crown parts of the underlying buds. Dense accumulations of inflammatory cells and exudate were noted in the tooth sacs. Inflammatory edema penetrated through the adjacent areas of the united epithelium, lifting it away from the enamel in a blisterlike fashion and destroying the epithelium.

This study was supplemented in 1935 by a report of a case, far more severe, involving the gangrenous deciduous mandibular left second molar of a 6-year-old boy who succumbed to pneumonia. The mandible was removed at autopsy. The microscopic findings differed from the first cases by the excessive breakdown of chronically inflamed tissue which replaced all the tissue between the roots of the deciduous tooth and the underlying bud. This resulted in the formation of numerous abscesses which brought about the destruction of the almost entire

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reduced epithelium of the bud. Highly infected granulation tissue resorbed an area of the exposed enamel and deposited a bonelike cementum into this cavity.

The present paper deals with the findings of the microscopic examination of two maxillas and one mandible of three children of the ages of 5, 5½, and 6 years, respectively. The children died of bronchopneumonia. The jaws were removed at autopsy and were roentgenographed.

After fixation and decalcification the parts presenting the changes of interest were cut out for the routine histologic preparation. Mesiodistal and buccolingual sections were stained with hematoxylin and eosin.

MICROSCOPIC PATHOLOGY

CASE 1.—The block contained the deciduous mandibular right molars and was sectioned in a mesiodistal direction (Fig. 1). The crown of the first deciduous molar had a deep cavity with a partially exposed and gangrenous pulp. The lateral parts of the cementum of the root showed a moderate resorption. The periapical area of both eroded roots was surrounded by a diffuse but dense round-cell infiltration within a highly vascular granulation tissue. Next to the root ends the infiltrated tissue had broken down, thereby forming small abscesses which communicated with the decomposed pulp. A rather moderate amount of inflammatory cells, mainly lymphocytes and plasma cells, were noted throughout the granulation tissue which replaced the interradicular periodontal membrane. This space was extremely widened at the expense of the bone between the deciduous molar and the underlying bud. The thinning of the coronal part of the bony crypt was mainly due to the osteoclastic activity of the granulation tissue rather than the pressure of the bud moving upward.

The continuity of the bony crypt was interrupted in two places corresponding to the extension of the periapical processes. It was here that the granulation tissue had broken through the bone, thus producing a broad communication between the periapical involvements and the tooth sac (Fig. 2). Some lymphocytes and plasma cells were noted in the sac tissue which exhibited an unusual fibrosis. A considerable fibrous wall bridged the wide gaps in the bony crypt opposite the apices (Fig. 2). This dense tissue must be regarded as the outer layer of the sac. Its excessive density must be regarded as the reaction to the adjacent inflammatory process. The rich network of capillaries, chiefly on the side of the proximal root next to the reduced enamel epithelium, appeared dilated and congested. Here, small areas of the enamel epithelium were missing and the exposed enamel was covered by exudate.

Of significance and importance for a thorough understanding of the inflammatory process, which took its origin from the infected pulp of a deciduous tooth, is the fact that the inflammatory changes traveled along the vessels into the bone marrow spaces below the affected tooth bud. Granulation tissue containing lymphocytes, plasma cells, and exudate not only occupied these areas but also extended far below the neighboring bud. This demonstrated clearly the diffuse nature of the infection. The pulp of the bud involved appeared affected by a passive congestion with dilated vessels, edema, and hydropic degeneration of the odontoblasts. There was a fairly broad zone of uncalcified dentine.

CASE 2.—The labiolingual section through the region of the deciduous maxillary central incisor of a 5-year-old boy shows the deciduous tooth and the bud of the permanent successor lying above.

The deciduous tooth was deeply decayed and its crown destroyed, the widened pulp canal contained remains of a pulp gangrene, and the apex communicated with a periapical abscess which was surrounded by a dense accumulation of inflammatory cells (Fig. 3). The palatal part of the alveolar process of the deciduous central incisor was missing and a dense fibrous tissue replaced the bone beneath the gingival margin. It contained isolated remains of the dental lamina and aggregations of inflammatory cells beneath the free gingival margin. The epithelial attachment on the palatal and labial side rested on the cementum, and its epithelium

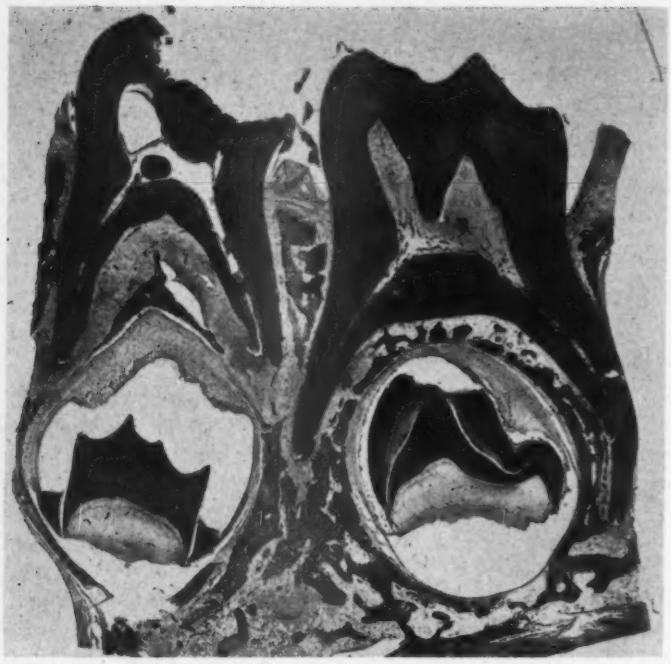


Fig. 1.—Mesiodistal section through the deciduous mandibular right molars and the underlying buds of their successors. Extensive occlusal caries in the first molar. Decomposed pulp, periapical inflammation with abscess formation on both roots. Inflammatory involvement of the tooth sac.



Fig. 2.—Higher magnification of Fig. 1, showing the distal root with abscess. Bone between root end and united enamel-epithelium of the bud destroyed. Strand of dense fibrous tissue separates periapical abscess from tooth sac.



Fig. 3.—Labiolingual section through the deciduous maxillary central incisor with its successor. Periapical abscess of the deciduous tooth in contact with the united enamel-epithelium of the bud.



Fig. 4.—Higher magnification of Fig. 3. United enamel-epithelium adjacent to the abscess transformed into a thin hyaline membrane.

sent papillae deep into the inflamed subepithelial tissue, closely following the course of the inflammatory cells. The gingival sulcus was deep.

On the labial side a part of the periodontal membrane was transformed into a vascular, heavily inflamed granulation tissue which had broken through the labial alveolar bone, thereby extending into the periosteum. Osteoclastic resorption, which resulted from the inflammatory condition, and bone deposition were noted on the alveolar bone. The marginal gingiva showed a dense small cell infiltration. The epithelial attachment proliferated along the cementum. There was a moderate gingival sulcus.

The abscess around the resorbed root end closely approximated the bud of the permanent successor, which was in its early prefunctional eruption-stage and lingual to the deciduous tooth. It was here that a thin strand of sac tissue intervened between the abscess and the united enamel epithelium of the bud (Fig. 4). In this area the united enamel epithelium was reduced to a membrane which in structure and stain appeared to be identical with the hyaline membrane along the enamel of embedded teeth observed and described by Bauer.[3] This short membrane was superimposed on and clearly discernible from the primary enamel cuticle. The rest of the sac tissue and the united epithelium did not reveal any involvement. Enamel, dentine, and pulp appeared intact.

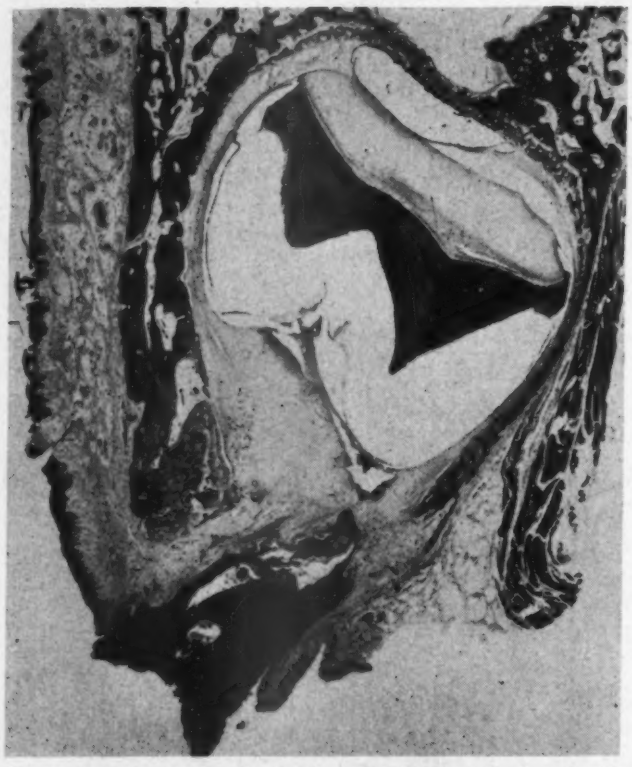


Fig. 5.—Labiolingual section through the buccal distal root of a deciduous maxillary left second molar with the bud of the permanent second premolar. Exacerbation of the chronic periapical inflammation of the deciduous tooth with abscess formation along the occlusal part of the enamel of the bud. Note the changes in the pulp of the bud and the edema in the tooth sac next to the pulp.

CASE 3.—Fig. 5 shows a labiolingual section through the region of the deciduous maxillary left second molar of a 5-year-old boy. The deeply decayed and resorbed root of this tooth was invested in a chronically inflamed granulation tissue with extensive hemorrhages and a papillary epithelial proliferation which covered the lingual side of the root and continued upward to the surface of the enamel of the bud of the permanent second bicuspid. The area around the root end demonstrated clearly an acute flare-up of the chronic inflammation (Fig. 5). Numerous polymorphonuclear leucocytes aggregated there and were also scattered through-

out the thick epithelial strands which lined a biconcave abscess. This abscess extended from the buccal cusp to the lingual cusp of the premolar bud and to the bottom of the central groove. The surrounding pyogenic membrane, which contained the proliferated epithelium, was crowded with polymorphonuclear leucocytes, and the adjacent sac tissue showed plasma cells, macrophages, and lymphocytes.

The united enamel epithelium appeared fairly well preserved along the lingual surface but failed to reach the top of the lingual cusp. All along the occlusal surface from the

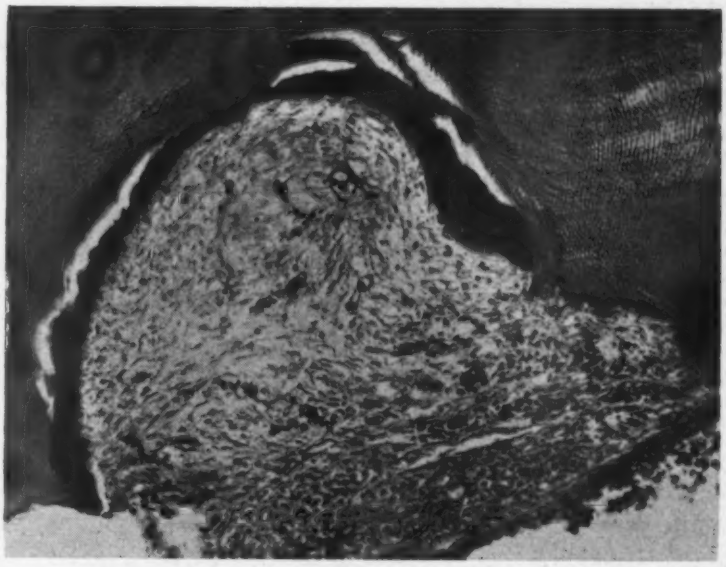


Fig. 6.—High magnification of Fig. 5, showing granulation tissue in a deep excavation of the enamel of the tooth bud. The resorption shows few Howship's lacunae to the right while the greater part of the surface is covered by a layer of deeply calcified cementum-like tissue.

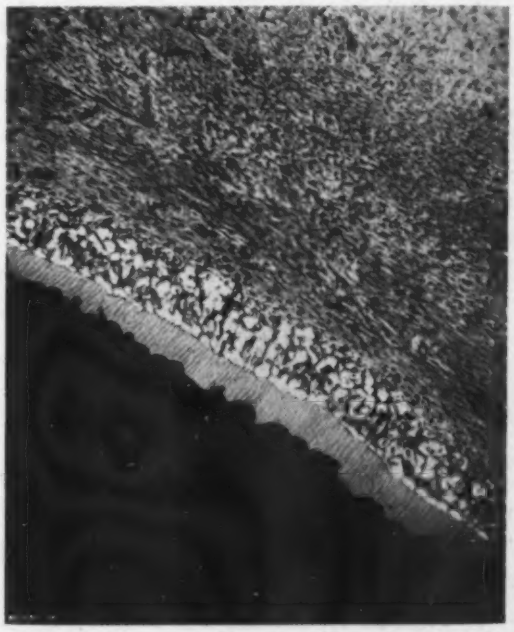


Fig. 7.—High magnification of Fig. 5, showing fibrosis of the pulp and hydropic degeneration of the odontoblasts of the tooth bud.

lingual cusp to the marginal border of the enamel of the buccal cusp, no typical united epithelium was detectable. Around the ends of the abscess the enamel was in direct contact with the granulation tissue, which had replaced the sac tissue and extended a short distance along the enamel. It was here that the enamel had undergone the most remarkable changes. In this area the surface of the enamel was found to be resorbed by granulation tissue which penetrated deep into the enamel. The resorption had come to a standstill, for one edge of the half-moon-shaped cavity showed definite Howship's lacunae in the enamel without ameloclasts (osteoclasts), while the remaining surface was covered by a bonelike cementum which was deposited on the enamel by the adjacent tissue (Fig. 6). This deeply calcified layer was the product of a metaplastic process of the granulation tissue. There was no osteoblastic activity, but the calcification of the adjacent connective tissue was apparent. The fact that this hard substance at some places had been torn from the enamel indicated that the connection between these two structures was not strong.

The inflammation proceeded farther into the part of the tooth sac next to the pulp and caused there an edema which appeared in large cystic cavities.

The pulp tissue close to the odontoblast layer consisted of very dense fibers that ran parallel to the dentinal surface. The odontoblasts underwent hydropic degeneration which stopped the deposition of dentine (Fig. 7).

DISCUSSION

These and the previous cases reported presented the opportunity to study the nature of the periapical involvement of deciduous teeth and the mode of extension to the buds of the pertinent permanent successors.

All instances exhibited acute exacerbation of chronic processes. The difference between the chronic periapical lesions of deciduous and permanent teeth became apparent. By far the largest number of chronic periapical lesions of permanent teeth shows the classic feature of chronic inflammation, i.e., proliferation of fixed cells, infiltration with lymphocytes and mononuclear cells, and the tendency to form a connective tissue wall which is usually connected with the periodontal membrane. However, the chronic periapical involvement of the deciduous teeth failed to demonstrate the attempt of the surrounding tissue to wall off the process. The acute process superimposed upon the proliferative and productive inflammation extended diffusely and rapidly into the neighboring areas and along the vessels into the distant parts around the buds of the permanent teeth.

This spread might be due to the lack of resistance of the tissue in which the deciduous teeth are invested and also favored by the pressure of the erupting bud of their successors. The pressure upon bone and soft tissue facilitated and amplified the extension of the inflammation into the sac of the buds.

The inflammatory reaction in the tooth sac affected gradually the enamel of the buds to various degrees. The sections employed did not present an early developmental stage of the bud with the stellate reticulum well preserved. The author believes that this avascular cushion would have effectively served as a protection to the enamel-forming cells or the enamel from the advancing granulation tissue. This assumption was confirmed by the findings around the tooth buds in congenital syphilis reported by Bauer.[4]

However, in the cases studied, the chronic periapical inflammation of the deciduous teeth met a later stage of tooth development which is characterized by the disappearance of the stellate reticulum and designated as the "prefunc-

tional phase of eruption" (Weinmann[5]). The united enamel epithelium became exposed to the approaching inflammatory process. The changes of this epithelial structure depended on the intensity of the inflammation and also the reaction of the sac tissue.

In one of the cases observed, the outer layer of the united enamel epithelium in the vicinity of the inflammation had been destroyed, and the remaining inner layer assumed the appearance of a thin, somewhat homogenous membrane, which showed here and there a definite affinity to eosin stain. It resembled very much the membrane which Bauer[3] found on the enamel of an embedded third molar as a result of an apical inflammation which proceeded from the gangrenous second molar. Since this membrane of the embedded tooth continued into the dental cuticle of the second molar and showed the same affinity to eosin stain, Bauer[3] concluded that the secondary enamel cuticle, which develops during the eruption as a product of the united epithelium when it unites with the oral mucosa, is the hyalinized and, occasionally, keratinized layer of the epithelial attachment. This transformation results from an inflammation often present in the surrounding oral mucosa during the shedding of the deciduous teeth.

Such a hyalinized and partially keratinized membrane formed upon the enamel of an erupting permanent tooth is likely to withstand the inflammatory action for some time.

However, in one of the cases observed, the inner layer of the united enamel epithelium was not able to undergo this helpful transformation into a protecting membrane in time. Here, an exudate had penetrated through the densified sac tissue and caused a hydropic degeneration of the epithelial cells. Yet no damage to the enamel substance was noted.

In another case granulation tissue had contacted the united enamel epithelium. In some areas the inflammatory process provoked a proliferation of the enamel epithelium to such an extent that strands of it joined the proliferated epithelium around the apex of the deciduous tooth, thus forming a network of epithelial strands which traversed the granulation tissue. In other areas the epithelium broke down, thereby exposing the enamel to the chronically inflamed tissue. The enamel appeared eroded, due to the activity of ameloclasts (osteoclasts) which produced a deep cavity. The large part of the lacunar surface of the enamel lesion was lined with a layer of an intensely and somewhat irregularly calcified cell-free substance which resembled primary cementum. This tissue had been deposited on the lacunar enamel without cellular activity, i.e., without cementoblasts or osteoblasts. It resulted from a metaplastic process which originated in the delicate connective tissue fibrils of the granulation tissue in the moon-shaped defect of the enamel. These fibrils differentiated into a homogenous substance, collagen, which fused together and took up calcium salts from the tissue fluid. This metaplastic cementum upon the corroded enamel is responsible for the yellowish, spotty areas seen in the so-called "Turner teeth." It is the partial replacement of resorbed enamel by a cementum-like tissue that makes up the essential feature of this developmental defect of certain permanent teeth.

The pathogenesis of these lesions of the enamel differs fundamentally from that observed in rickets and other systemic diseases. These last-mentioned conditions bring about the destruction of the ameloblasts, so that the enamel formation discontinues without being replaced by another hard substance.

The conclusions drawn from these microscopic studies of human material were verified by the following clinical observation which was based upon a reliable clinical record given by the mother of the young patient.



Fig. 8.—Photograph of the teeth of a boy, 12 years of age, showing disfiguration of the maxillary right permanent incisors. Erosions of the enamel covered with a yellowish bonelike substance. Roentgenogram inset into the right upper corner.

CLINICOPATHOLOGICAL RELATION

A boy, 12 years of age, came to the clinic accompanied by his mother. When he was 4 years old, the deciduous maxillary right central and lateral incisors were decayed, and they broke down when he reached the age of 5 years. At this time a painful swelling developed above these teeth and persisted for about five days. The swelling had been lanced. The remains of these two deciduous teeth were retained unduly long in the maxilla and were finally removed by a dentist. Their successors emerged later in the mouth, and when they appeared, both teeth were shorter than the left incisors. The mesial marginal portion of the labial surface of the central incisor was occupied by a rough, nodular, yellowish substance of hard consistency, which extended beneath the inflamed, flabby marginal gingiva. There was not the slightest doubt that this lesion, irregularly bordered by normal enamel, was not caries. Small spots of yellowish material were also deposited on excavations of the incisal edge and the distal surface. The lateral incisor exhibited less significant changes. The incisal edge showed a shallow, irregular erosion. Here, too, the grooves were speckled with small spots of a hard, yellowish substance. (Fig. 8.)

The roentgenograms revealed a shorter, slender, and irregularly outlined central incisor. There was a translucent area in its mesiolabial part that extended lingually toward the

distal side. The coronal part of the pulp and two apical thirds of the pulp canal contained a radiopaque tissue. The roentgenogram of the lateral incisor did not demonstrate a particular feature.

The mother of the boy denied permission to remove the disfigured central incisor but agreed to the removal of a small splinter from the yellowish, hard tissue for microscopic examination. This substance proved to be a bonelike cementum with lacunae and canaliculi.

SUMMARY

In conclusion, it may be stated that this pathological-clinical study proves that the so-called "Turner teeth" result from periapical inflammatory processes of deciduous teeth. These processes extend toward and affect the buds of the pertinent permanent teeth during their prefunctional stage of eruption. Yet, the possibility exists that mechanical injuries of the latter may produce similar lesions.

The examination of autopsy material containing deciduous teeth and their successors showed that periapical processes of deciduous teeth failed to stimulate the development of a fibrous wall which could localize the lesions. They spread diffusely through the bone marrow toward and around the buds of the successors, thereby affecting the important protective layer of the young enamel, the united enamel epithelium. Various changes of the latter were observed.

In mild cases this epithelial structure in the area next to the apical process of the deciduous tooth appeared to be reduced to a flat, hyalinized, somewhat keratinized, membrane, which the author believes to be similar to, if not identical with, the secondary or dental cuticle.

In some instances the inflammation stimulated the enamel epithelium to proliferate.

In other cases the united enamel epithelium was destroyed, thereby exposing the enamel to inflammatory edema and granulation tissue. The former was found to be harmless to the enamel, while the latter eroded the enamel and deposited on the surface of the deep excavation a well-calcified metaplastic cementum-like substance.

A clinical case substantiates the above conclusions.

The removal of deciduous teeth with periapical involvement should be the method of choice.

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SPONTANEOUS REGENERATION OF BONE FOLLOWING EXCISION OF SECTION OF THE MANDIBLE

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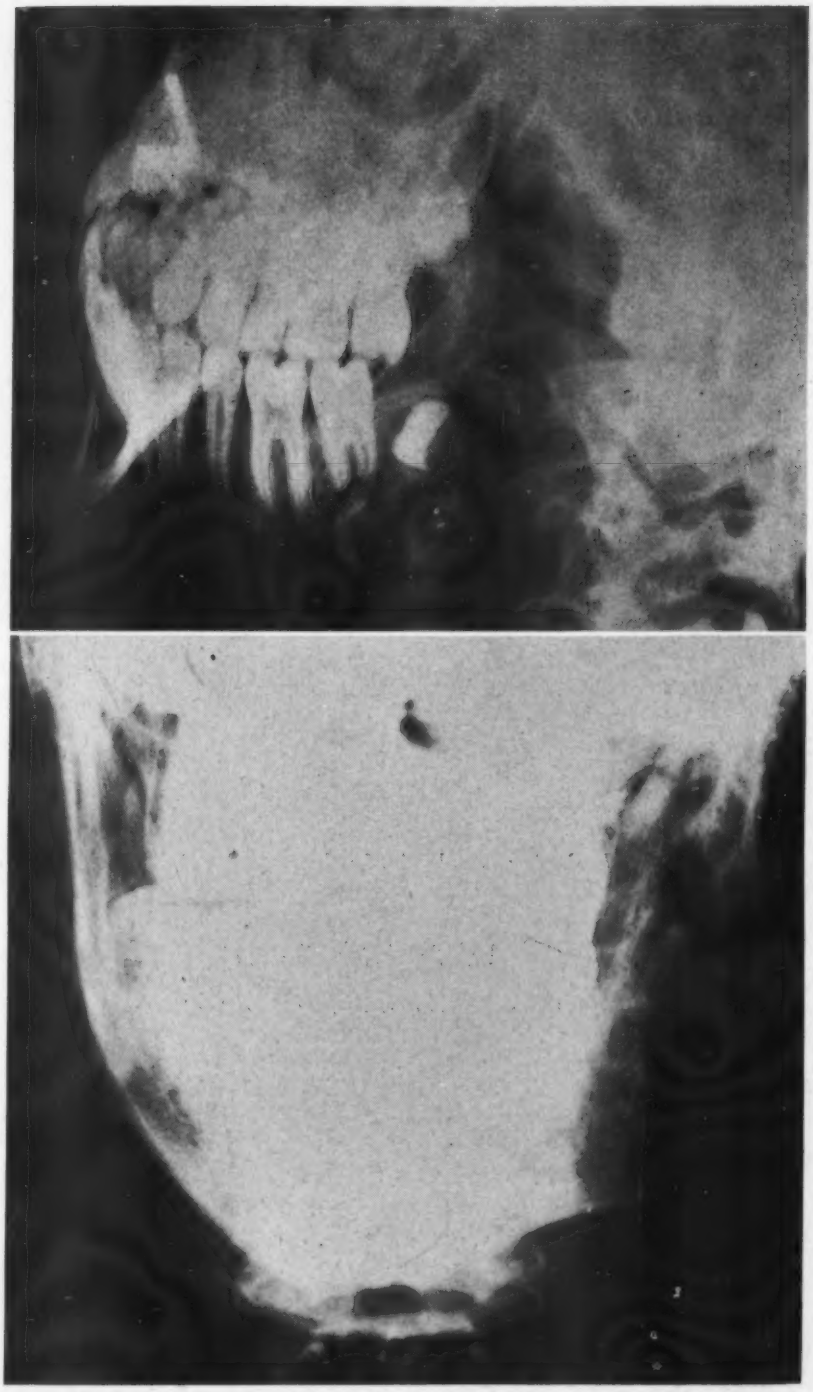
THE patient, a 10-year-old white boy, was first seen in August, 1935. There was an indurated, painless swelling of the left side of the mandible over the angle and ramus of the jaw. The x-ray examination revealed a large cystic tumor involving the left side of the mandible, and having the appearance of an adamantinoma. A differential diagnosis from giant-cell tumor was made (Fig. 1).

An operation under avertin and ether anesthesia was performed. Exposure of the anterior surface of the tumor was obtained through an incision made laterally in front of the canine tooth along the gingival margin toward the intermaxillary space. The mucoperiosteum was dissected free on the buccal side of the jaw, and the thin bony shell covering the cystic cavity was removed with mallet and chisel. The cavity extended as far as the middle of the ramus posterosuperiorly, and as far as the canine region anteriorly. The walls, while almost bare, contained soft spongy bone which was easily curetted out. A section of soft tissue near the anterior wall was sent to the laboratory for an immediate diagnosis, but a positive one could not be made. Therefore, it was considered advisable to curette the entire cavity as much as possible. The constant oozing of blood from the walls of the tumor mass was controlled by packing the cavity with bone wax.

The subsequent pathologic report was giant-cell tumor. The specimen consisted of dense connective tissue and a number of irregular trabeculae of bone and bone formation from osteoid tissue. A number of giant cells of the benign type were seen. There was no definite evidence of malignancy.

The patient made an uneventful recovery following the operation. X-rays were taken at intervals for comparison, and in April, 1936, they showed considerable new bone formation, but the bulk of the ramus showed no great diminution in size. The patient continued to report for observation every six months for the next three years. Resection of the jaw was advised, but the parents were inclined to take a waiting attitude.

When the patient was examined in December, 1939, operative treatment was decided upon. Fig. 2 shows his appearance before the second operation. Splints, which were to be used immediately following the operation, were constructed. The appliance (Fig. 3) consisted of an upper and lower wire splint fastened to the teeth. The splint carried two buttons, one at the upper second molar region and the other at the lower premolar region. A bar supplied with a prong at each end extended from the upper button to the lower one. When in position, the ends of the prongs were slightly pressed together, thus making a loop around the button. As seen in the diagram, this bar extending from the upper to the lower jaw was made in two sections, the upper part being a tube, while the lower end was a threaded wire. By turning the screw at the lower end, the bar could be graduated to the length necessary to give good occlusion of the teeth. As the mouth was opened, there was no disturbance of the lateral movements of the jaw due to the fact that the loop at each end of the bar fitted loosely around the button. When the teeth were in contact, this bar forced the lower jaw into normal occlusion.



A.

B.

Fig. 1.—Preoperative x-rays showing the extent of the lesion involving the mandible; *A*, lateral view; *B*, anteroposterior view.



Fig. 2.—Photograph showing the patient's appearance before the second operation.

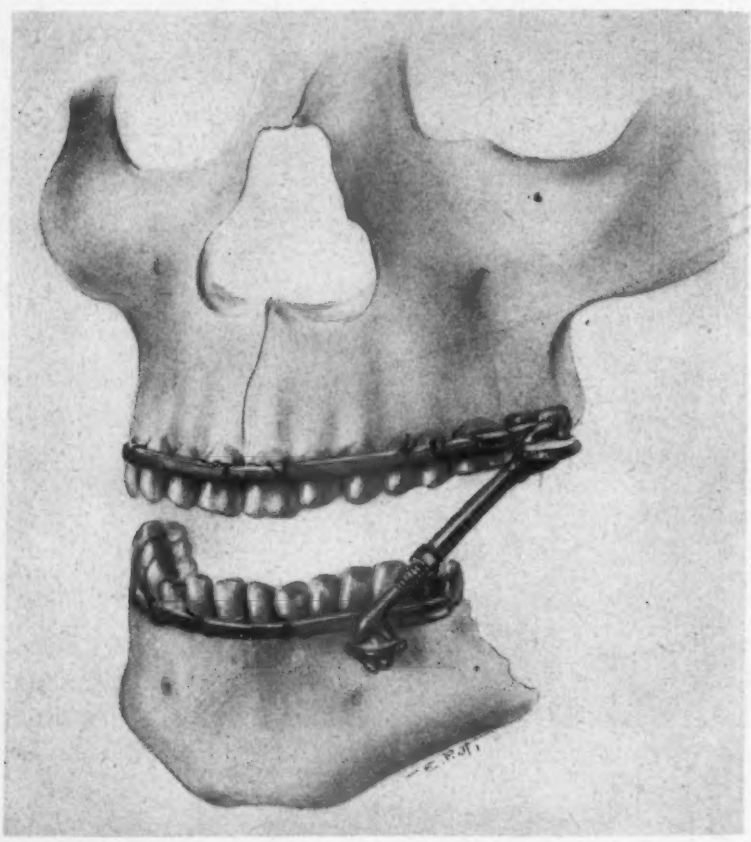


Fig. 3.—Diagram of appliance (see description of appliance in text).

The patient was operated on in January, 1940, under intratracheal ether anesthesia. The lower left first molar tooth was extracted, and the mucoperiosteum was dissected from the mandible on the inner side of the jaw. Through a stab wound in the face, a Gigli saw was passed around the mandible at the site of the tooth extraction, and the mandible was cut through at this point. Blunt dissection was carried out until the mandible was exposed on both sides up to just below the sigmoid notch. Eventually the mandible was cut across completely at that level, and the specimen removed. It seemed that the resection was beyond the limits of the growth. Suturing and drainage were carried out, and the splints previously prepared were attached to hold the anterior fragment of the mandible in correct occlusion.

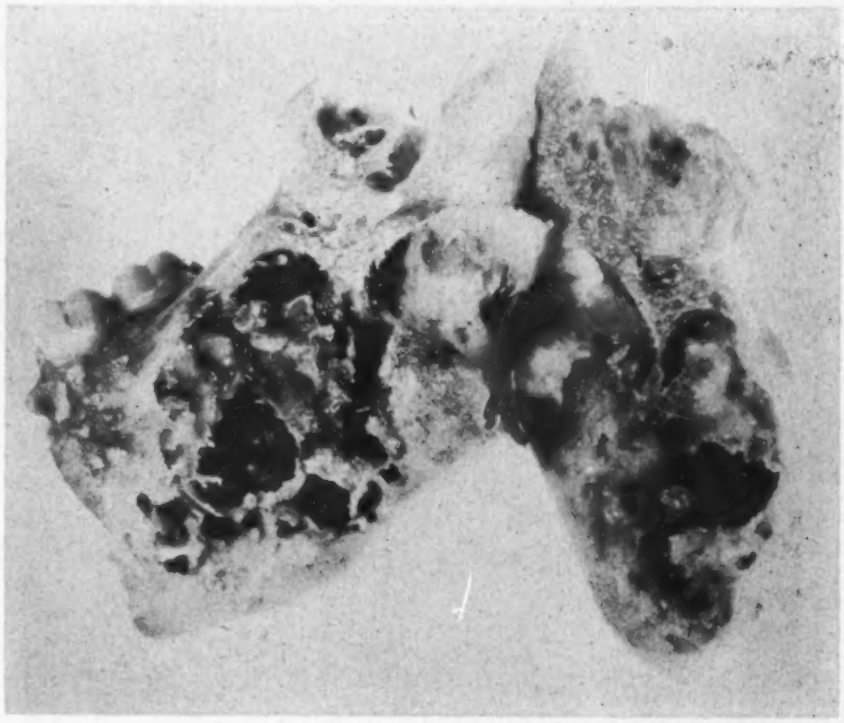


Fig. 4.—Photograph showing the gross appearance of the sectioned specimen.

Fig. 4 shows the gross appearance of the sectioned mass. The cut surface of the specimen presented a multilocular cystic appearance within the cancellous bone. The larger cyst spaces measured about 2 cm. in diameter. These spaces were not lined with cystic membrane as would be found in adamantinoma. Instead, the surfaces consisted of smooth, dense bone. Examination of the specimen microscopically revealed a rather dense fibrous connective tissue stroma composed, for the most part, of spindle-shaped cells and containing numerous irregularly shaped trabeculae of bone (Fig. 5). These trabeculae were newly formed; in some places they were homogeneous, and in others contained irregular lamellar lines. Numerous multinucleated giant cells were in close apposition to the bone spicules, and in some areas were free in the stroma. Where the bone appeared older, the trabeculae were rimmed by an eosinophilic layer of osteoid, which in turn was bordered by a regular layer of osteoblasts. Many focal areas of calcification were seen in the stroma in which newly formed blood vessels were noted. In one area a large deposit of hemosiderin was seen, and adjacent to this were numerous multinucleated giant cells. On the periphery of the lesion adult



Fig. 5.—Photomicrograph showing bone trabeculae and dense fibrous connective tissue stroma.



Fig. 6.—X-ray showing the appliance in place and the bony regeneration.

bone was found, and the marrow spaces were fatty in nature with some hematopoietic tissue. Diagnosis: Ossifying fibroma.

The patient made an uneventful recovery, and was seen frequently for readjustment of the retaining appliance which held the occlusion in good position. Postoperative x-rays taken in April, 1940, showed regeneration of bone. Fig. 6 shows the appliance in place and the bony regeneration. This process eliminated the necessity for bone transplantation. When the patient was seen six weeks later the appliance was removed from the teeth, and the occlusion was quite satisfactory. There was no appreciable external deformity.



Fig. 7.—Postoperative appearance of the patient without the appliance.

COMMENT

Conflicting theories concerning the ossifying fibroma have been published. Some investigators believe it to be a tumor of medullary origin, while others consider the onset of the lesion to be associated with trauma and hence not a true tumor. The latter regard these lesions in the same light as giant-cell tumors. The ossifying fibroma is a slow-growing, nonpainful lesion, said to be more frequent in females in the second and third decades of life. The usual symptom is asymmetry of the face or a disturbance of the occlusion. Two types are recognized: the hyperostotic and the hypo-ostotic. However, these are the same lesions but seen at different stages of development. The hyperostotic type shows a cellular fibrous tissue in which are deposited newly formed trabeculae of bone; simultaneously there is evidence of bone resorption, but to a much lesser degree. In the hypo-ostotic type the marrow spaces are widened, and the bony trabeculae are undergoing resorption. New bone formation is absent, and the tissue is made up of a loose connective tissue in which many blood vessels are found.

The case presented is of interest because the clinical and radiographic evidence pointed to a diagnosis of adamantinoma. Before a radical operation was performed, however, it was deemed advisable to determine the true nature of the lesion by means of a biopsy examination. The report was "giant-cell tumor." Continued observation within the period of three years did not show

any appreciable change in the size of the tumor; on the contrary, x-rays taken repeatedly showed some regeneration of bone.

The second point to emphasize in this case is that the resection of the bone was made possible by stripping the periosteum. This, undoubtedly, explains the marked regeneration of bone which took place within six months. Moreover, the mobility of the mandible did not seem to prevent regeneration.

The retention appliance used in this case helped to hold the remaining part of the mandible in its proper occlusal relationship to the upper teeth, so that the patient was able to masticate while healing. In addition, it gave such good fixation that, when the large gap had filled in with bone, the teeth were still in good position. When the appliance was removed, the patient had an excellent functional and cosmetic result (Fig. 7).

475 COMMONWEALTH AVENUE

Case Reports

CASE NO. 95

FATAL INFECTION FOLLOWING TOOTH EXTRACTION: GANGRENOUS STOMATITIS

B. J. Masuda, D.D.S., M.S., A.B., Philadelphia, Pa.

THE patient, an 8-year-old white girl, complained of sore gums and slight swelling following the extraction of an acutely infected upper right first molar tooth.

She was immediately admitted to the hospital and two days later the gingival area became red and tender, with pain in the face, ear, and neck. The ulceration extended to the buccal mucosa and the face became swollen and indurated. Later an area of redness appeared on the outside surface of the cheek. This became brawny, then black, then a necrotic plug sloughed out leaving a perforation in the cheek and a condition of gangrene (Fig. 1).

Necrosis spread rapidly in the bone of the maxilla and the teeth lost their bony support and were exfoliated. Six days after onset of gangrene and two months after onset of swelling, the patient developed streptococcus septicemia, and prostration followed, with delirium and death (Fig. 2).

DISCUSSION

Gangrenous stomatitis, also known as cancrum oris or noma, is a fulminating gangrene of the mouth and an advanced stage of ulcerative stomatitis. Fusospirochetal disease (Vincent's infection), may also take the form of gangrenous stomatitis.[1] It usually attacks the gums or cheeks and involves a considerable portion of the face. It is a progressive disease associated with marked destruction, and the prognosis therefore is unfavorable as it has a high mortality rate.

Etiology.—This rare disease is commonly found in undernourished, debilitated young patients, between the ages of 4 and 11 years, and usually accompanies such diseases as scarlet fever, measles, dietary deficiency disease, whooping cough, or pneumonia. A specific cause has not been demonstrated.

Davis, Pilot, Weaver, and Tunicliff claim that noma is due to the blocking of the circulation by the invasion of the tissue with the fusiform bacillus and spirochetes of Vincent's in one stage or another of its cycle. Others claim that other organisms such as the staphylococci, various types of streptococci and anaerobic organisms may be involved. It begins as a small sensitive nodule or ulcer on the gingiva, the walls of which become jagged as it widens. The ulceration progresses rapidly and followed by an indurated swelling in the cheek with a more or less red area appearing on the skin. Pronounced salivation and fetid odor are usually present. The disease then perforates the cheek. The patient has definite signs of toxemia from the start and often goes into a coma toward the end.

In the case reported the process was essentially that of gangrene. Suppurative inflammation was probably the result of a secondary infection. Microscopic smears and cultures from the lesion revealed the presence of the following organisms: (1) Hemolytic streptococci. (2) Fusiform bacilli. (3) Spirilla of Vincent. It is not known, however, whether

Read before the Medical Staff of the Mercy Hospital, Philadelphia, Pa., May, 1945.



Fig. 1.—The patient, an 8-year-old white girl, complained of sore gums following the removal of an acutely infected upper right first molar tooth. Six days later, the face became swollen, indurated, and brawny in color. A necrotic plug sloughed out, leaving a perforation in the cheek and a condition of gangrene. This shows the patient the second day after the onset of gangrene.



Fig. 2.—Necrosis spread in the bone of the maxilla and the teeth lost their bony support and were exfoliated. Streptococcus septicemia developed and prostration followed, with delirium and death. This shows the patient six days after onset of gangrene and two months after onset of swelling.

these were the etiological factors or just incidental contaminants. Very often blood dyscrasias were found to be the underlying cause of this disease.

Salama[2] reported a fatal case in which noma occurred in a man 20 years old, as a result of severe anemia. The disease in many cases is ushered in through the extraction of teeth, especially unerupted third molars, which could very often be the cause. Palmer and Carr[3] reported such a case occurring in a man 55 years old. The disease in this case started after the extraction of an acutely infected upper right first molar tooth.

Diagnosis and Clinical Characteristics.—From the onset of the disease, the following manifestations were noted: (1) Waxy induration of the skin. (2) Greenish-black necrosis and crusting of the affected area. (3) A definite zone of demarcation. (4) A rapid extension of the disease accompanied by foul odor. (5) Increased salivation. (6) Enlargement of the superficial, deep facial, and cervical lymph glands. (7) boardlike infiltration. (8) Temperature ranging from 103° to 106° F. (9) Prostration followed by delirium.

Prognosis.—The prognosis in this case as in others was unfavorable, because the disease has a high mortality rate (from 80 to 90 per cent).

Treatment.—General treatment, that is, building up the patient's resistance through rest, nourishment, and blood transfusions is, in my opinion of the greatest importance. These were instituted in this case. Early surgical interference by endothermy and electrocoagulation were also performed. As the disease spread rapidly, radical treatment was instituted early; however, with no success.

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1930 Chestnut Street

CASE NO. 96

DERMOID CYST IN THE FLOOR OF THE MOUTH

B. J. Masuda, D.D.S., M.S., A.B., Philadelphia, Pa.

THE patient, a man, 24 years of age, presented a dermoid cyst of sublingual origin. He came to the outpatient department of the dental dispensary of the hospital thinking that his condition was associated with a dental infection. He complained of difficulty in chewing and swallowing, and upon clinical, radiographic, and case history examinations, a diagnosis of a dermoid cyst of sublingual origin was made. He stated that he first noticed the growth about five years before, after which time it developed to a slowly growing painless mass (Fig. 1). On palpation, the cystic area was smooth and round as a hard-boiled egg, and of a putty to a soft rubber consistency. The prognosis was favorable. Treatment consisted of complete excision and enucleation of the cyst with its fibrous capsule under a local anesthetic. The patient made a good recovery.



Fig. 1.—A dermoid cyst of sublingual origin in a 24-year-old man. It is of five years' duration. Though congenital, these tumors are usually not observed until early adult life. It was a slow-growing painless mass, and the patient first noticed it when he experienced discomfort and difficulty in chewing and swallowing.

DISCUSSION

Etiology.—Dermoid cysts are of congenital origin and, although they are present at birth, they do not become evident until early adult life. Usually they appear between the ages of 10 and 25 years. In this case, the patient was 24 years of age. This cyst was composed of ectodermal structure and was believed to be caused by the inclusion of the epidermis, with tissues formed from the mesoderm.

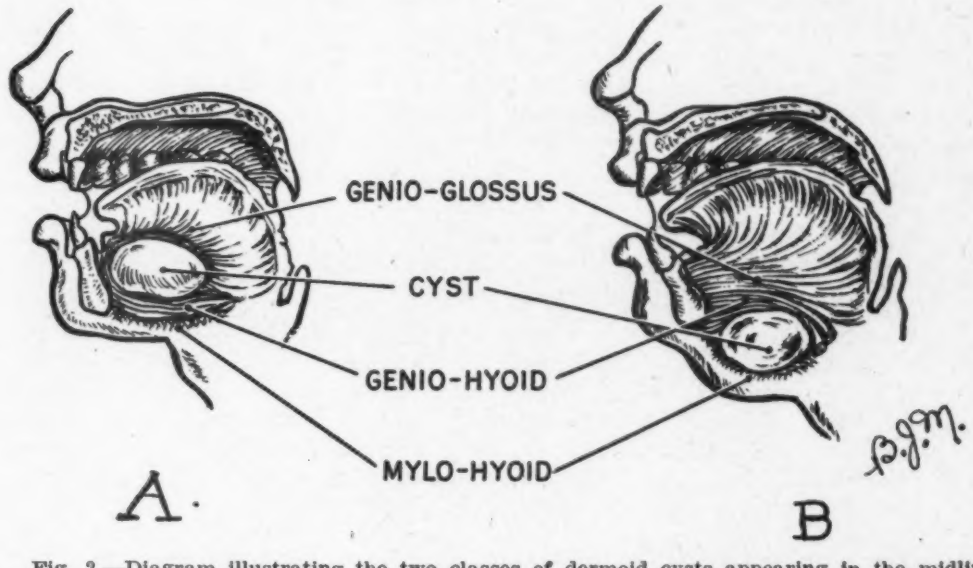


Fig. 2.—Diagram illustrating the two classes of dermoid cysts appearing in the midline behind the chin: *A*, Above the mylohyoid muscle, that is, between the genioglossus and geniohyoid muscles. *B*, Below the mylohyoid muscle, that is, between it and the geniohyoid muscle.



Fig. 3.—This dermoid cyst was round in size, 51 mm. long by 46 mm. wide. It was of a putty to a soft rubber consistency, pitted on pressure, and was whitish-yellow in color.

Clinical and Microscopic Examinations.—These cysts form a slowly growing, painless mass, and sometimes have been found to contain hair, teeth, sweat glands, and semisolid material of a cheesy consistency. They vary in size from a golf ball to a tennis ball. In some cases their growth is so extensive as to cause dyspnea (difficulty in breathing), dysphagia (difficulty in swallowing) and dysphonia (difficulty in talking). These also were the complaints of this patient. Although the swelling is painless, pain may result sometimes from injury or irritation of the cyst. Microscopic examination of the cystic wall in the reported case showed that it was made up of stratified epithelial lining surrounded by dense connective tissue (Fig. 6).



Fig. 4.—A ranula or retention salivary cyst also appears in the same location as a dermoid cyst, but varies from it by its bluish-gray color (dermoid cysts are usually whitish-yellow), clear viscid content, and tendency to fluctuate on palpation. Differential diagnosis of these cysts could, therefore, be ascertained by aspiration and the examination of the contents. Compare with Fig. 1.

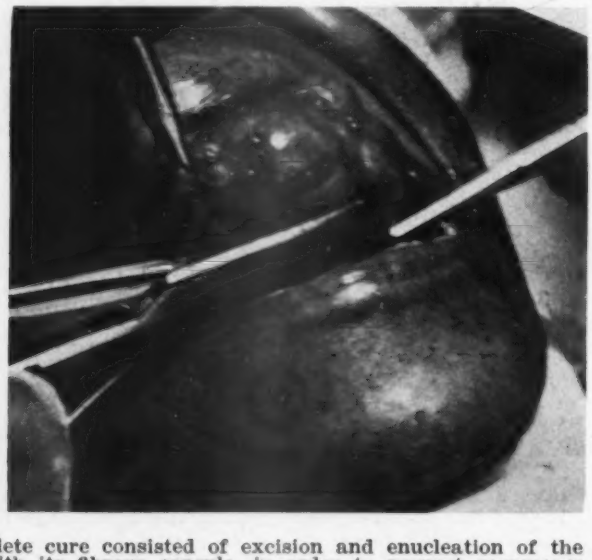


Fig. 5.—Complete cure consisted of excision and enucleation of the dermoid cyst in its entirety, together with its fibrous capsule, in order to prevent recurrence. A local anesthetic was the one of choice in this case and has proved satisfactory in this operation.

Occurrence and Classification.—Dermoid cysts are usually situated in the midline and sometimes laterally around the angle of the jaw. The latter are believed to have shifted from a central midline location.[1] Marshall[2] reported cases of dermoid cysts which occurred in the ears. In this case, the cyst occurred above the mylohyoid muscle.

Dermoid cysts can be classified according to their anatomical location, as follows:

1. Cysts above the mylohyoid muscle, that is, between the genioglossus and geniohyoid muscles forming a bulge beneath and pushing the tongue upward (Fig. 2, *A*).

2. Cysts below the mylohyoid muscle, that is, between the mylohyoid and the geniohyoid muscle. They form a swelling in the submental region (Fig. 2, *B*[3]).

Diagnosis.—Important diagnostic features that aid in the diagnosis of these cysts and differentiation from other cysts are their location, color, and firmness, and their contents.

Dermoid cysts of sublingual origin are whitish-yellow in color (Fig. 1), while a ranula or a retention salivary cyst, which also appears in the same location, is bluish-gray. Likewise, a dermoid cyst is of a soft rubberlike consistency, may pit on pressure, and contains a semisolid cheesy material (Fig. 3). On the other hand, a ranula fluctuates and secretes a viscid clear fluid (Fig. 4).

The differential diagnosis of these cysts can be made by aspiration and also by means of x-ray, which sometimes reveals the presence of teeth, hair, and sweat glands in dermoid cysts.



Fig. 6.—It contained a semisolid cheesy material. Microscopic examination of the cystic wall showed that it was made up of stratified epithelial lining, surrounded by dense connective tissue.

Prognosis.—The prognosis is favorable, especially if the epithelial wall is removed in its entirety to prevent recurrence (Figs. 3 and 5).

Treatment.—Complete excision and enucleation of the dermoid cyst is the only method of cure. Since dermoid cysts are encapsulated by a fibrous capsule lined by squamous epithelium, the structure must be removed entirely in order to prevent recurrence. The surgical procedure for the enucleation of the dermoid cyst varies with the type of cyst. If the cyst is above the mylohyoid muscle, such as in this case, a free incision of the mucous membrane is made in the sublingual region, and by blunt dissection the entire cyst is removed. The free ends of the mucous membrane are then sutured into place (Fig. 5).

If the cyst, on the other hand, is situated below the mylohyoid muscle, an incision is made in the submental region through the skin and subcutaneous tissues in order to get free access and a clear view of it. This is done by careful blunt dissection of the median raphe of the mylohyoid muscle. Care must be exercised in removing the entire capsule with its epithelial lining to prevent recurrence. After the cyst is removed, the external incision is closed by suture.

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1930 CHESTNUT STREET

Editorial

Oral Surgery Comes of Age

THE establishment of a National Board of Oral Surgery, strongly backed by the American Society of Oral Surgeons, is a milestone in the history of our profession. The need for such a board has been felt by many for a considerable number of years, and the certification of qualified oral surgeons will be a great aid to hospitals and educational institutions in the selection of men for appointments to their staffs. The board will also set standards for the educational program of future applicants. It will determine the type of training for undergraduate and postgraduate students. Postgraduate study will doubtless include additional instruction in the basic medical sciences, especially the general principles of surgery, oral pathology, and surgical anatomy of the face and neck. Two years of internship in an approved teaching hospital should give the student the practical experience in clinical, roentgen, and pathologic diagnosis of diseases of the mouth and jaws, in anesthesia and oral surgical techniques, including the general preoperative and postoperative care of the patient.

The oral surgery clinic should be situated in a hospital. Performing oral surgical operations on patients in an outpatient dental school clinic should be a thing of the past. It is as outdated a procedure as taking out tonsils in a physician's office. With the exception of exodontia and minor dentoalveolar surgery in individuals who are in good health, oral surgical operations should be performed only on hospitalized patients. Complicated fractures of the jaws, osteomyelitis, extensive cysts and tumors, odontogenic neck infections, to mention a few, require bed rest, general medication such as chemotherapy or penicillin administration, and the type of supportive treatment that can be given only in a hospital. In addition, general anesthesia for any prolonged surgical procedure should certainly not be given to the patient who, so to speak, comes in from the street. After the operation he may not observe, or often forgets, instructions which frequently are inadequate, and a stay in the so-called recovery room is no substitute for the good general care a surgical patient receives in a hospital.

The undergraduate students should be given an opportunity to visit this oral surgical teaching clinic, so that they can observe the methods of diagnosis and the treatment of oral surgical patients in a hospital. They should also be required to attend the dental conferences, which should be held at regular intervals.

The postgraduate student, on the other hand, during his internship should serve in rotation in the outpatient dental department and in the house. In the former he will gain experience in the diagnosis of oral diseases, in the

extraction of teeth, and other minor procedures. In the latter, he will assist the oral surgeon at operations until he is able to operate under supervision, a staff member assisting him. He should attend clinics, conferences, ward rounds, and autopsies to get as much general training as possible. He should take careful histories of oral surgical patients, attend consultations that will assist him in appraising the type of anesthesia that is indicated, and the general treatment the patient should receive. He should be in charge of the after-care both while the patient is on the ward and later when he is an outpatient.

Thus, the man with basic dental training will be able to supplement his knowledge not only by acquiring techniques of oral surgical procedures, but also by familiarizing himself with the general physiologic and physiotherapeutic principles that should govern the care of all surgical patients.

K. H. T.

MAY, 1946

Oral Surgery

including

Oral Medicine, Pathology, Diagnosis, and Anesthesia

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THE CLINIC OF THE MASSACHUSETTS GENERAL HOSPITAL AND THE HARVARD SCHOOL OF DENTAL MEDICINE

Volume VI

KURT H. THOMA, D.M.D.,* HOWARD W. SMITH, D.M.D.,† HAROLD F. BOSCO, D.M.D.,† FRED R. BLUMENTHAL, D.M.D.,‡ AND HENRY M. GOLDMAN, D.M.D.§

ANKYLOSIS OF THE MANDIBULAR JOINT

KURT H. THOMA, D.M.D.

ANKYLOSIS of the mandibular joint is a serious disease since it not only interferes with the mastication of food and nutrition, but also prevents hygienic measures, prophylactic care, and treatment of dental caries, so that patients with this ailment generally suffer from extensive multiple caries and periapical infection. Furthermore, if contracted early in life, the lack of function will prevent normal development of the jaw. This in turn prevents normal eruption of the teeth and causes a disfigurement which handicaps the patient in many ways.

It is curious that patients with ankylosis make every attempt to take care of their teeth; they may even have all the teeth extracted under the most difficult circumstances instead of having the cause of their trouble removed. An operation will restore the free movement of the jaw so that dentistry may be performed to save the teeth. The following are arguments for early operation in cases of ankylosis:

1. In young patients it prevents underdevelopment of the jaw and resulting difficulties with dentition and disfigurement (mandibular retrusion and bird face [Figs. 530 and 531], as well as maxillary protrusion and contraction due to the use of dilators to force the jaws apart).

2. In patients of any age it prevents atrophy and fibrosis of the muscles of the jaw caused by long-continued inability to masticate, handicapping the patient after operation and requiring prolonged physiotherapy.

3. In patients of any age it keeps caries from becoming rampant. Since the control and treatment of caries are difficult when ankylosis is present, the loss of

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many teeth, which makes the balancing of the jaw difficult after operation, especially in bilateral cases, is prevented.

ETIOLOGIC FACTORS

The causes of ankylosis are many. The report includes a study of eighteen patients which have been seen with this affliction, thirteen with unilateral, five with bilateral involvement. Of these, sixteen came to operation. Table I shows a classification of the cases according to the etiology, involvement, and type of operation performed.

TABLE I. ANKYLOSIS OF THE MANDIBULAR JOINT

CAUSE	NO. OF PATIENTS	INVOLVEMENT		OPERATION	
		UNILATERAL	BILATERAL	CONDYLECTOMY	OSTEOARTHROTOMY
Birth injury	2	2	0	0	1
Rheumatoid arthritis	6	4	2	2	6
Marie Strümpell's disease	2	0	2	0	4
Septic arthritis	2	1	1	0	3
Osteomyelitis	2	2	0	0	1
Malunion of condylar fracture	4	4	0	3	1
Total	18	13	5	5	16

Birth trauma of the joint occurs in cases of difficult delivery, particularly forceps delivery. Often other bones are fractured as well. One of the two patients seen had a birth weight of 14 pounds, and during a breech delivery the wrist and left leg were fractured. The injury of the jaw was not discovered until later when it was noticed that he could open his jaw only slightly. This is a common experience in this type of case. Sometimes the injury is associated with a deformity of the external ear (Fig. 530). Often it goes unnoticed until the child gets his deciduous teeth or attempts to masticate food. A result of early ankylosis is underdevelopment of the jaw, since growth of the mandible is greatly influenced by muscular function, as pointed out in a previous paper (Thoma, 1938).* This results in a so-called bird face (Fig. 531) and irregular dentition with impaction of teeth.

Rheumatoid arthritis generally has a clear history with involvement of other joints. The cause of the disease is not well known. Infection seems to be an important factor, but the disease is not due to a specific organism. Predisposing causes play an important role, such as fatigue, trauma, exposure, constitutional infirmities, and emotional factors. Often it follows debilitating diseases of the infectious type, such as pneumonia, scarlet fever, rheumatic fever, etc.

In the acute stage of the disease the joints are generally swollen and hot; there is a tendency of the disease to migrate from joint to joint. The onset is characterized by fever which may last for weeks. Later there is destruction of the cartilage of the joint including the meniscus, followed by erosion of the bony surfaces. Adhesions form with periarticular thickening and thickening

*Thoma, K. H.: Principal Factors Controlling Development of Mandible and Maxilla, AM. J. ORTHODONTICS AND ORAL SURG. 24: 171, 1938.

of the capsule. The head and neck of the condyle become greatly enlarged, obliterating the mandibular notch, and sometimes the coronoid process is extremely narrow. The joint finally becomes entirely destroyed and ankylosis results. Atrophy of the muscles generally accompanies the disease. If contracted early in life, as in the first case reported below (Case 113), underdevelopment of the mandible results, as in the case of ankylosis caused by birth trauma.



Fig. 530.



Fig. 531.

Fig. 530.—Ankylosis of the jaw caused by birth trauma injury to the mandibular joint and external ear, and resulting in underdevelopment of the jaw (Courtesy of Harvard Dental School).

Fig. 531.—Bird face caused by birth trauma injury of the mandibular joint and resulting in ankylosis.

Marie Strümpell's disease is a special form of rheumatoid arthritis in which the spine gradually stiffens. The rigidity increases until the entire spine becomes ankylosed, preventing movements of the back and neck. In some cases the hip joints, and more rarely the mandibular joints, become ankylosed as well. Table I includes one case in which there was bilateral involvement of the mandibular joint alone, another in which the hip joints and mandibular joints both were involved. The treatment of the latter patient has been reported in Case 82 in the Clinic of the Massachusetts General Hospital, Volume IV, published in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, April, 1945.

Suppurative arthritis may be caused by extension of an infection of the middle ear. The second case (Case 114) reported below is of a patient presenting ankylosis following acute mastoiditis associated with fracture through the base of the skull. Osteomyelitis of the ramus and condyle may extend into the joint of the mandible, but in many cases the joint does not become involved, limitation of motion being due to condylar enlargement. Suppurative arthritis is also said to be caused by secondary gonorrheal infection, although we have not seen a case, and by syphilis. Hematogenous infection was observed in a patient who

developed bilateral mandibular anklyosis following general streptococcic septicemia from a cut on a finger. This case is also of interest because it is the only one in which wound sepsis developed on both sides after the operation.

Septic arthritis may be differentiated from the rheumatoid type by the monoarticular involvement with a history of neighboring infection. In the hematogenous type the focus may be present and multiple involvement may occur. Patients complain of great pain and tenderness; there is heat, swelling, edema, muscular trismus, and, later, suppuration with fever, leucocytosis, and sweats, with ankylosis the final result.

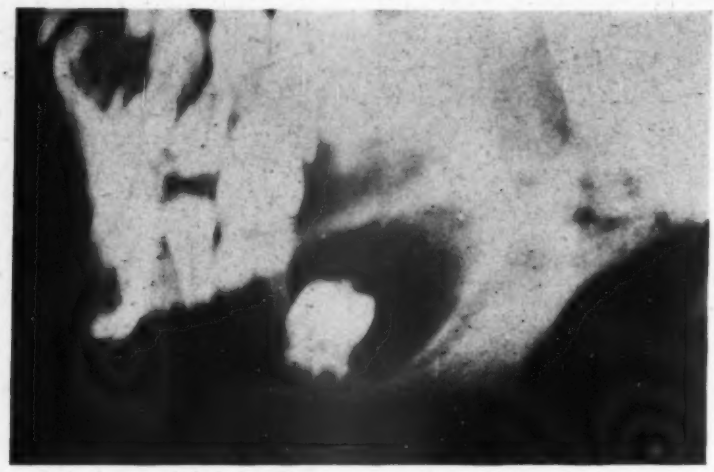


Fig. 532.—X-ray of the jaw of a patient with generalized osteomyelitis showing enlargement of the condyle.

Osteomyelitis may affect the mandibular condyle without involving the joint itself. It is the deformity of the condyle in these cases which causes limitation of motion besides the associated muscular trismus and periarticular swelling. Suppuration often causes fistulas, and permanent fistulas result from incision and drainage. The cause of osteomyelitis of the condyle is frequently an extension of an abscess of the pterygoid fossa; the infection travels along the external pterygoid muscle to the condylar neck. A patient who had an abscess in the pterygoid fossa from an infected maxillary molar, which extended to the ramus causing osteomyelitis of the vertical ramus, was reported in the Clinic of the Massachusetts General Hospital, Volume III, Case 55. Another patient with a similar abscess in the pterygoid fossa causing osteomyelitis of the head of the condyle will be reported below.

Osteomyelitis also may develop through hematogenous channels. A previously reported case* serves as an example. The patient, a 10-year-old boy with a history of abscess and multiple osteomyelitis involving the tibia, humerus, and ulna due to streptococcic septicemia, developed a swelling over the mandibular ramus with adenopathy, which was due to osteomyelitis extending into the condyle. This caused limited opening of the jaw. The condyle became

*Thoma, K. H.: A Case of Generalized Osteomyelitis With Interesting Jaw Involvement, Am. J. Orthodontics and Oral Surg. 22: 516, 1936.

greatly enlarged and showed characteristic osteolytic areas (Fig. 532). Later ankylosis resulted. An x-ray taken nine years later showed an enlarged irregularly deformed condyle (Fig. 533).

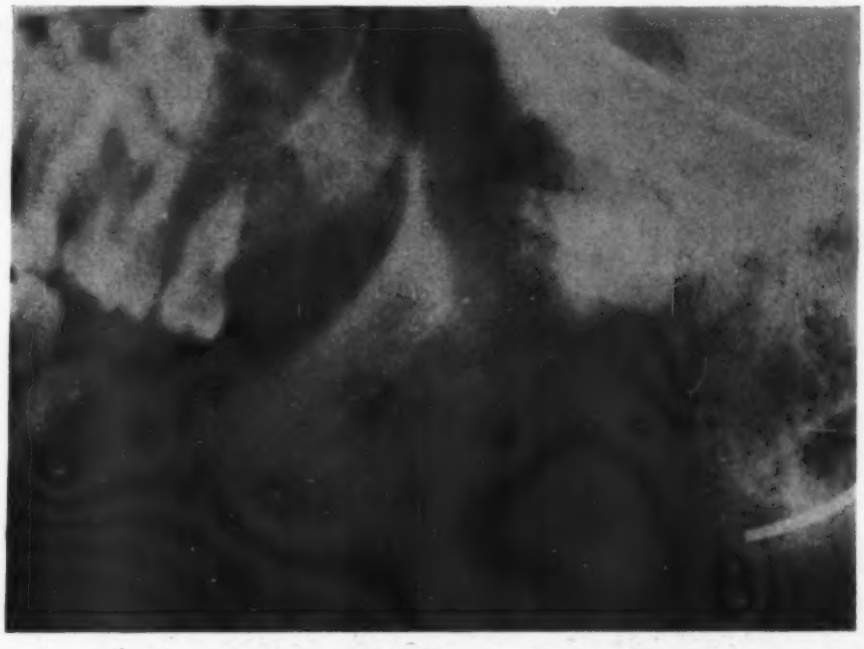


Fig. 533.—X-ray of the jaw of the same patient nine years later showing ankylosis of the condyle.

Fractures of the condyle, untreated or treated ineffectively, may cause limitation of motion. The most common fractures causing ankylosis are comminuted fractures of the head of the condyle, as well as fractures of the tympanic plate. In both instances there is hemorrhage into the joint space leading to fibrous, and later, bony union. There is usually a history of bleeding from the ear at the time of the accident. Comminution of the condyle may also result in synarthrosis, as in Case 94 reported in the Clinic of the Massachusetts General Hospital, Volume V.

In cases of badly displaced condylar fractures or fracture dislocations, improper reduction will cause traumatic arthritis with pain on motion and limitation of lateral and forward movement of the jaw. Two such cases have been reported in the Clinic of the Massachusetts General Hospital, Volume V, Cases 95 and 96.

CLINICAL MANIFESTATIONS

The clinical manifestations of ankylosis, the final result of the disease processes discussed, vary somewhat. Inability to open the jaw and difficulty in masticating food are the outstanding symptoms. In most instances the immobilization develops gradually, and even when completely ankylosed, the patient may be able to move the jaw slightly. This motion does not necessarily take place in the joint; it may be due to the elasticity of bone or to movement in the cranial sutures. If the ankylosis is unilateral, some motion is made pos-

sible by the unaffected joint, especially in a vertical direction. This can be observed by examining the patient's posterior occlusion, an observation which sometimes is valuable in making a differential diagnosis between unilateral and bilateral ankylosis.

In cases in which the disease was contracted early in life, underdevelopment of the mandible, malocclusion, and impaction of unerupted teeth are characteristic signs. The receding jaw with a narrow protruding maxilla, caused by the use of dilators and exercises to counteract the freezing of the joint, produces a bird face, as shown in Fig. 531.

Pain is not an outstanding symptom; it is present only in acute suppurative disease and in traumatic arthritis caused by malunion of condylar fractures.

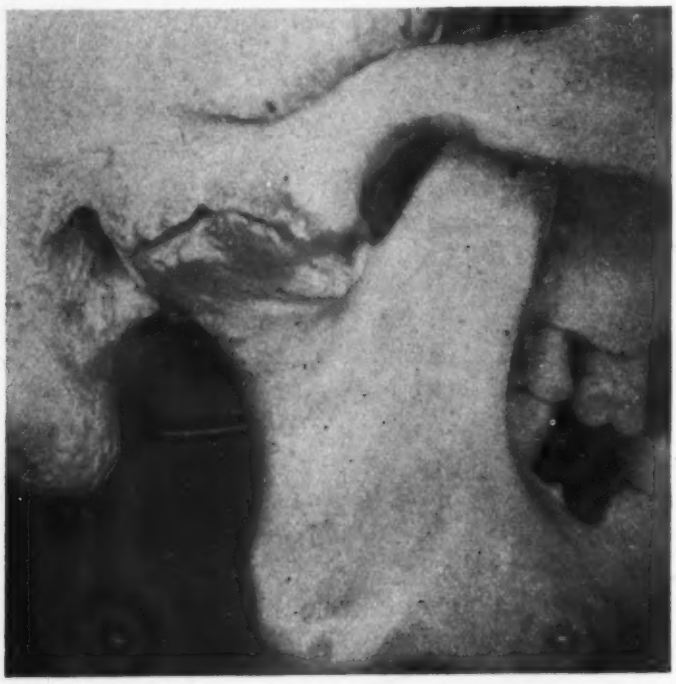


Fig. 534.—Skull showing characteristic changes caused by temporomandibular ankylosis due to rheumatoid arthritis. (Courtesy Army Medical Museum, Washington, D. C.)

The deformity of the mandible in adults is limited to the condyle and the joint. In children, as pointed out, marked micrognathia results. With this is associated a prominent angle of the jaw, and a curve of the inferior border which may denote an attempt at bending the bone by the powerful depressor muscles attached to the symphysis, which come into function when great force is needed to open the jaw. The characteristic appearance of an ankylosed joint is shown in Fig. 534, a skull from the Army Medical Museum. It shows the short, wide neck and the flat, fused joint, the outer lip of which can be detected, in most instances, after the capsule is removed at operation.

ROENTGEN EXAMINATION

Good x-rays are of great importance. If there is any question as to which side is ankylosed, or whether there is any motion possible, temporomandibular

exposures should be made in forced open and closed positions of the jaw. The lateral view, however, gives more important information. It shows the width of the neck of the condyle, and gives a picture of the situation as it will be presented at the operation. The anteroposterior view also is of great importance since it shows the lateral enlargement of the condyle and the amount of hyperostosis or exostosis present. In order to be able to remove the entire bony mass which is attached to the base of the skull without injury to important structures, it is necessary to know how far it extends medially.

OPERATIVE PROCEDURES

Operative procedures are of three types: arthroplasty, condylectomy, and osteoarthrotomy. Dilators and exercisers are of very little use; they cause more harm than good. In young individuals they deform the maxilla and frequently the mandible, and injure the teeth.

Arthroplasty is an operation which removes a section of bone at the neck of the condyle. Temporal fascia, fascia lata, or muscle tissue is interposed to produce a false joint. Occasionally the insertion of foreign material has been recommended to form a new joint in order to prevent union, which may occur in spite of everything, after this type of operation.

Condylectomy is advised in all cases in which the joint has started to become ankylosed, or in which arthritic processes cause pain and limitation of motion. The operation consists of an osteotomy at the neck of the condyle, after which the condyle is excised by detaching the capsule while a chisel is inserted into the joint space to sever any adhesions that may be present.

Osteoarthrotomy is used if there is complete bony union. After the osteotomy has been completed, an osteotome is inserted at the site of the former joint and the entire condylar mass is removed from the mandibular fossa of the temporal bone with blows from a mallet.

In both condylectomy and osteoarthrotomy, a bone cavity of considerable size is formed which will prevent reattachment of the ramus at the amputated end. This cavity will fill in with tissue, but in order to prevent a hematoma from forming, a rubber-dam drain should be placed into it for twenty-four hours. In septic cases a rubber tube, such as a piece of No. 10 catheter, is inserted through the fistulous opening, if present, into the wound for the injection of a weak solution of penicillin (250 to 500 units per cubic centimeter). The incision should be closed with a subcuticular suture for the best cosmetic results. This operation should make it possible for the patient to open and close the jaws. Displacement is prevented by the accessory ligaments and vertical position is maintained by the occlusion of the teeth. The only interference with a good result is muscular atrophy and fibrosis, which is generally present in cases of long standing; this can be overcome to some extent.

Case 113

Unilateral Mandibular Ankylosis due to Rheumatoid Arthritis

L. B. (515066), a 25-year-old man, was admitted to the hospital on Dec. 21, 1945, with a diagnosis of ankylosis of the jaws due to rheumatoid arthritis.

At the age of 3 years the patient had had pneumonia, and gradually from then on his joints began to lose mobility. The temporomandibular joints had slowly become ankylosed. Both elbows, knees, ankles, and wrists also have limited motion. He has been under the care of his local physician, and was referred for treatment of the ankylosis. Examination showed an arthritic patient with motion of the jaws limited to 1 cm. vertically.

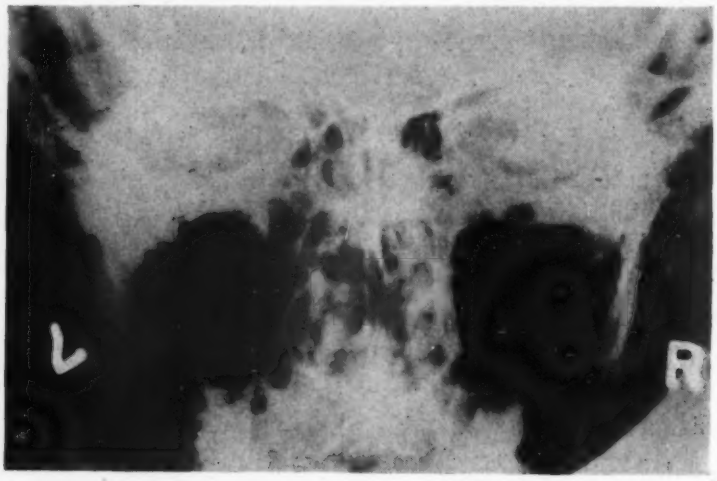


Fig. 535.—Case 113. Anteroposterior x-ray showing a very much enlarged condyle on the left.

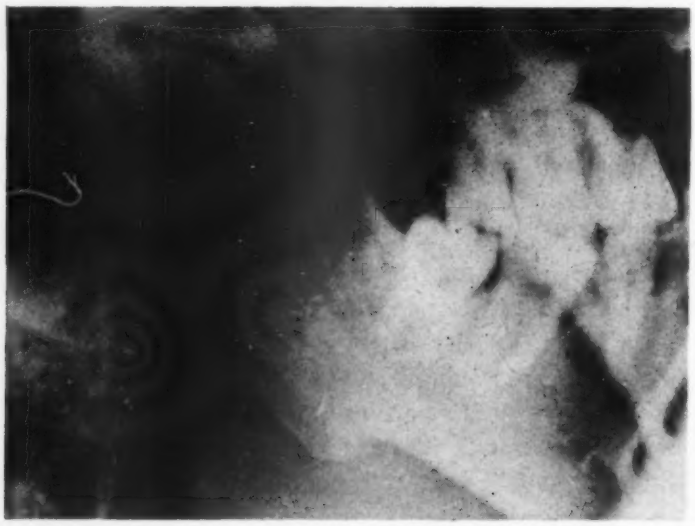


Fig. 536.—Case 113. Lateral view showing the enlarged condyle with an irregular and partly obliterated joint space. Note calcified stylomandibular ligament.

X-ray examination in the anteroposterior view showed a very much enlarged condyle on the left (Fig. 535). In the lateral view an irregularly widened condyle was seen with an irregular and partly obliterated joint space (Fig. 536). The stylomandibular ligament was calcified and there was a notch at the inferior border of the mandible at the junction of the ascending and horizontal rami.

On December 22 under intratracheal gas, oxygen, and ether anesthesia, an osteoarthrotomy was performed. The face was prepared in the preauricular and temporal regions, and a vertical, angulated incision was made in front of the ear, extending as far as the attachment of the lobe. The subcutaneous tissue was divided and the temporal fascia and zygomatic

arch exposed. From here the dissection was carried deeper. The periosteum of the zygomatic arch over the neck of the condyle was incised, as well as the capsule of the joint. The joint space was found to be obliterated by fibrous and osseous structures, and there was very little motion in it. The condylar retractors were inserted, and the bone divided at the neck of the condyle from the mandibular notch backward in a horizontal line. The osteotomy was performed by means of drills and osteotomes. After it was completed, an osteotome was inserted into the joint space to detach the condyle from the base of the skull. The remainder of the condyle was detached in this manner, but when the wound was inspected, it was found that another large piece of bone was attached to the base of the skull medially to the glenoid fossa. This was also detached with osteotomes. The amputated end of the ramus was made smooth with bone files. There was no excessive bleeding as all bleeding vessels were tied with catgut. The subcutaneous tissue was closed with interrupted catgut sutures. A very small drain was inserted through the subcutaneous tissue into the space created by the removal of the bone, and the skin was closed with a subcuticular suture.

The patient received 24,000 units pencillin intramuscularly on the day of operation, and 96,000 units on each of the next three days. The wound healed uneventfully; the x-ray examination on December 27 showed that the left condyle had been amputated. The patient went home on December 28, to be followed at the office. The sufure was removed on the tenth postoperative day, and the patient was discharged, relieved, ten days later.

Case 114

Unilateral Mandibular Ankylosis due to Mastoiditis in Case of Fracture of the Base of the Skull

R. D., a 6-year-old boy, was referred for examination because his head had ached several times during the previous week, and because he was unable to open his jaws and eat properly. The illness started eighteen months before when he was knocked unconscious by an automobile; he never before had been injured and had had no previous trouble with his ears or jaw. He had been treated at a hospital for fracture of the left femur. Soon after admission to the hospital he had developed an acute mastoiditis for which he was operated upon. A definite fracture of the skull, not visible in the x-ray, was discovered at operation. He was dismissed two months after the accident and was followed in the outpatient department of the same hospital, the last visit being ten months ago.

Physical examination showed a thin, pale, poorly nourished child, who was unable to open his jaw (Fig. 538). The right side was absolutely fixed; on the left he had vertical motion of about 2.5 mm. The mandible was slightly retruding.

Roentgen examination showed a normal mandibular joint on the left and bony ankylosis and hyperostosis of the condyle on the right. The mandible was slightly underdeveloped, the second molar developing posterior to its normal position (Fig. 537).

Diagnosis: Unilateral bony ankylosis of the right mandibular joint. Osteoarthrotomy was advised.

Under avertin and ether anesthesia at the Brooks Hospital, an incision was made in front of the ear, extending as far as the attachment of the lobe. The subcutaneous tissue was divided and the zygomatic arch exposed. Then the posterior attachment of the masseter muscle to the zygomatic arch was severed. This brought into view the exostosed bone attached to the head of the condyle. When the capsule was opened, the articular surface was found to be solidly ankylosed to the glenoid fossa. An osteotomy was performed at the neck of the condyle. The jaw then was forcefully opened and became free and movable. Next, the enlarged condyle, which was the size of a pigeon egg, was excised, being separated from the base of the skull with a chisel. The end of the masseter muscle was sutured to the internal pterygoid over the amputated surface of the ramus. A rubber dam drain was inserted to prevent a hematoma from forming. The fascia was sutured with catgut and the skin with horsehair.

The immediate postoperative condition was good, and the next day when the patient was examined, it was found that he could open the jaw wide and without pain (Fig. 539). There was some local swelling about the wound, but no infection. The drain was removed on the second day. On the fourth day the patient developed a temperature of 103.5° F., without pain in the jaw or other symptoms, and on the sixth day the temperature was 104.5° F. At this time a diagnosis of acute follicular tonsillitis was made by a consultant. The boy received treatment, and the healing progressed uneventfully from then on. He was given chewing gum to exercise his muscles, and when dismissed was much improved physically, owing to the fact that he ate well and could masticate his food. At a re-examination two months later, it was found that he was eating well and without any discomfort.

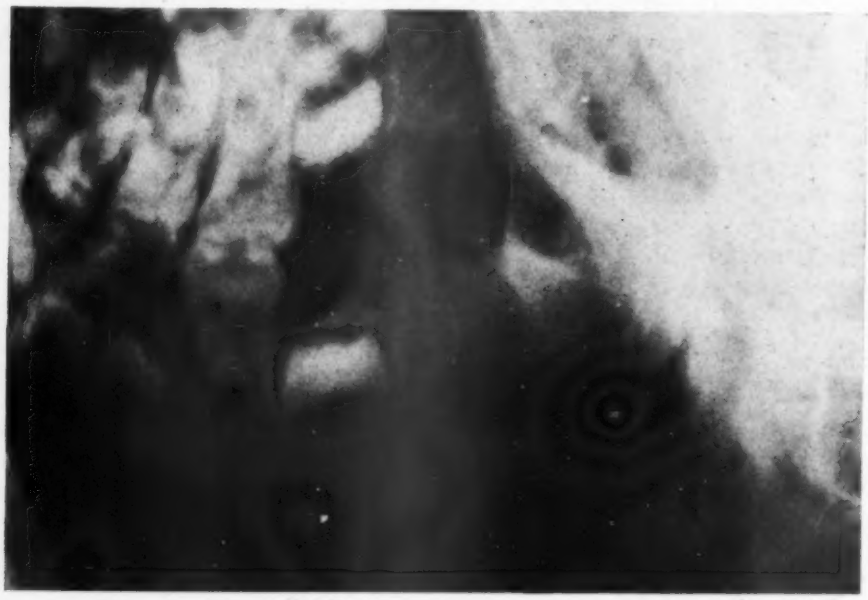


Fig. 537.—Case 114. X-ray of boy 6 years old showing ankylosis and hyperostosis of the right condyle.

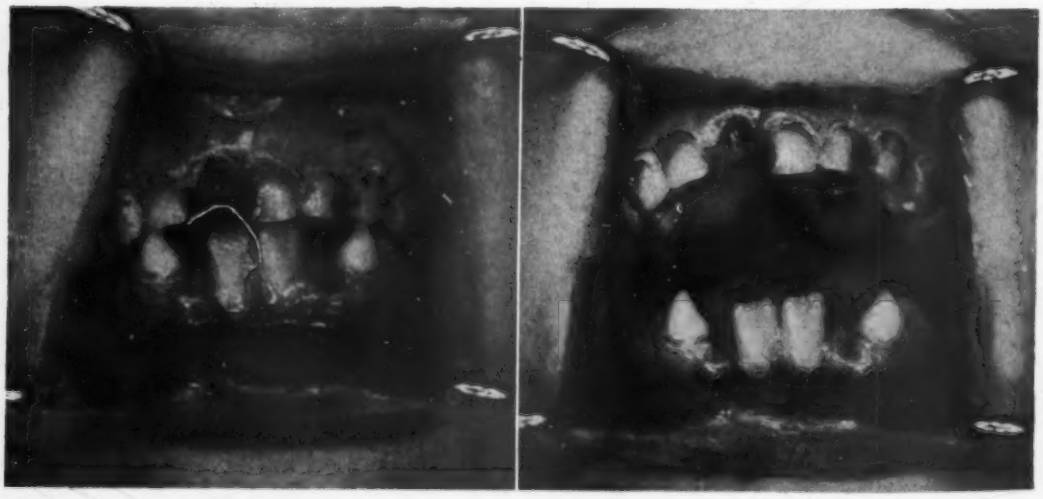


Fig. 538. Fig. 539.

Fig. 538.—Case 114. Ankylosis of the jaw before operation.
Fig. 539.—Case 114. Photograph showing the extent to which the patient could open the jaws on the second postoperative day.

Case 115

Unilateral Partial Ankylosis Caused by Osteomyelitis of the Condyle

A. S. (512318), a 28-year-old woman, was admitted to the hospital on Nov. 26, 1945, with a diagnosis of ankylosis of the left temporomandibular joint and osteomyelitis.

One year before the patient had had an upper left molar extracted. Following this she experienced chills, and during the next week a swelling anterior and inferior to the left ear and trismus developed. One month after the extraction there was little improvement. The patient was admitted to an outside hospital, and incision and drainage was performed. About one-half cup of pus was evacuated anterior to the ear. This relieved the trismus so that she could open the mouth about 1¾ inches. Another incision and drainage was performed two months later, and four months after this the patient was readmitted to the hospital and received penicillin (75,000 units every three hours) for nine days. One month later another incision and drainage was performed. She had made monthly visits to her dentist for treatment. Three weeks ago, while visiting in this city, the patient was referred to our clinic.

Examination showed moderate swelling anterior to the left ear, with two draining sinuses and three scars (Fig. 540). The mouth could be opened only 1¼ inches.

X-ray examination showed a definite osteolytic area extending from the external surface of the condylar neck into the head of the condyle (Fig. 541). The condyle appeared to be enlarged. A diagnosis of osteomyelitis of the condyle was made.

On November 27, under endotracheal gas, oxygen, and ether anesthesia, a condylectomy was performed on the left side of the mandible. An angulated vertical incision was made in front of the left ear. The subcutaneous tissue was divided and the head of the condyle dissected down upon. The capsule was incised and the periosteum of the condylar neck, and the bone laid bare. A probe was inserted into the upper fistula in the skin which was anterior to the incision, and this led directly to the head of the condyle where an opening could be seen which led into the marrow cavity of the condyle. Since there seemed to be no infected bone below the neck of the condyle, it was decided to remove it by performing an osteotomy below this tract. This was done with burs and osteotomes. The capsule was completely severed, but due to the peculiar enlargement of the condyle, considerable difficulty was encountered in removing it from the glenoid cavity. After the condyle finally was removed, there was some bleeding, which was arrested with fibrin foam; the foam was removed after the hemorrhage stopped because it was felt that no foreign body should be left in the infected wound. A probe was then inserted into the fistula at the angle of the jaw, and this led directly from beneath the jaw into the cavity made by the amputated condyle. Therefore, it was felt that probably both fistulas drained the same area. No sequestrum or diseased bone could be found when the rest of the ramus was examined. A piece of No. 10 soft rubber catheter was then inserted into each of the two fistulas as far as the amputation wound, and attached to the skin with dermalon sutures. The incision was closed with interrupted sutures in the subcutaneous tissue, and a subcuticular suture in the skin (Fig. 542). Pressure bandages were applied. During the operation, the patient received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water, containing 18,000 units of penicillin.

The patient was given 72,000 units of penicillin intramuscularly on the day of operation, and 192,000 units on each of the next ten days. Penicillin instillations into the wound, 500 units per cubic centimeter every four hours, were started and continued for five days.

X-ray examination on November 29 in anteroposterior view showed that the left condyle had been excised. There was some thickening of the ascending ramus, probably due to an old osteomyelitis. No definite sequestra or cavities were seen. The right side was normal (Fig. 543).

On the third postoperative day the patient was comfortable and the wound was healing well. There was no drainage from the sinuses. The lower jaw could be opened wide without pain. There was some deflection to the left.

On the sixth postoperative day the catheters were removed, and the mouth sprayed out. The patient was put on a soft solid diet, and given jaw exercises. The mouth could be opened at least 1½ inches. The wound healed uneventfully, and the patient was discharged on December 8, to be followed at the office. The suture was removed December 9, and examination showed all the sinuses completely closed.

Pathologic examination: A piece of extremely dense bone measuring 3 by 2.5 by 1.5 cm. with muscle fibers attached was submitted; also a smaller piece of cancellous bone with a grayish central portion. Microscopic examination showed a large section of bone con-

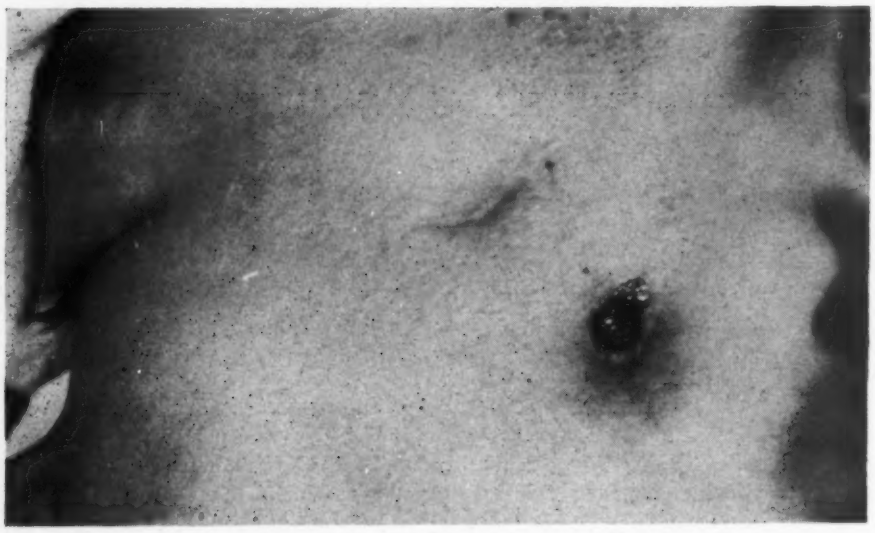


Fig. 540.—Case 115. Photograph of patient with osteomyelitis of the condyle showing an active fistula and healed wound where incision for drainage was made.

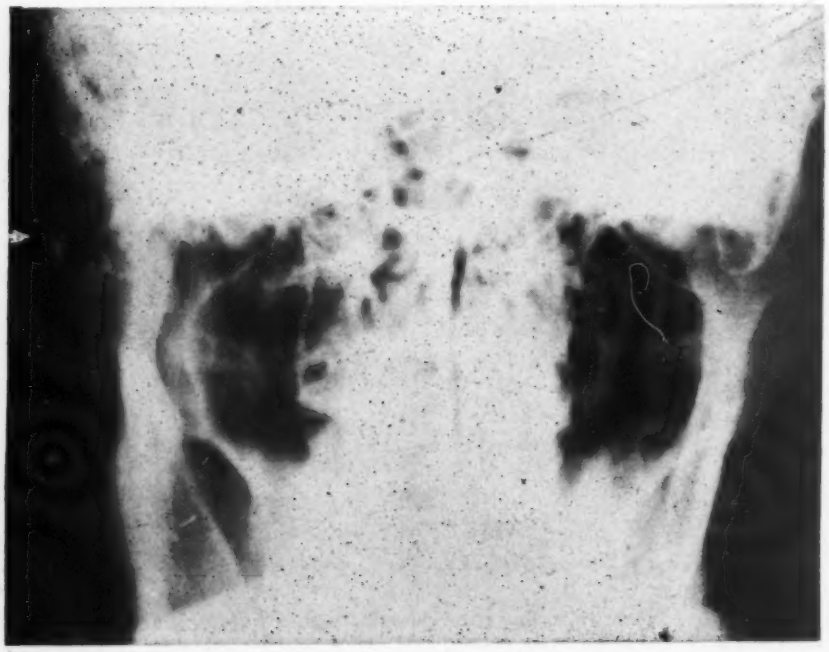


Fig. 541.—Case 115. X-ray of the left condyle showing bone destruction due to osteomyelitis.



Fig. 542.—Case 115. Postoperative photograph showing incision for condylectomy closed with subcutaneous sutures, and catheters inserted into the preauricular fistula and the fistula below the angle of the jaw for the local administration of penicillin.

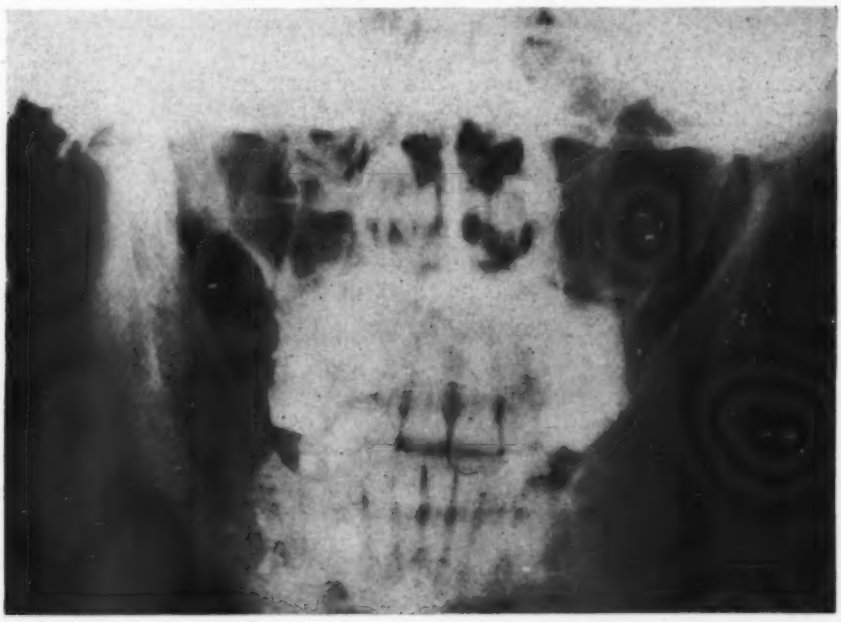


Fig. 543.—Case 115. Postoperative x-ray showing condyle excised and catheters inserted for the local administration of penicillin.

sisting of dense, thick, anastomosing trabeculae. The bone appeared viable for the most part, the bone cells being present. The marrow spaces were filled with fatty marrow. Some hemopoietic tissue was in evidence. In one section the marrow spaces were fibrotic and contained inflammatory cells, chiefly plasma cells and lymphocytes. In another section the marrow spaces were filled with débris, red blood cells, and a few areas of inflammatory cells. In this area the bone appeared nonviable, the bone cells being absent (Fig. 544). Cuplike resorptive areas on the edges of some of the trabeculae were noted. Diagnosis: Osteomyelitis.

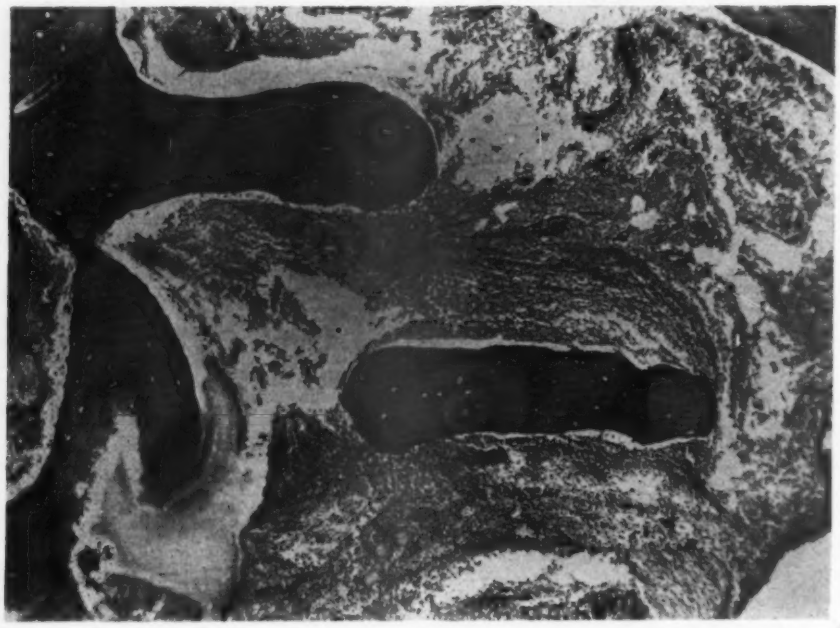


Fig. 544.—Case 115. Photomicrograph of the central portion of the excised condyle showing chronic osteomyelitis.

SUMMARY

Eighteen cases of ankylosis of the temporomandibular joint have been analyzed as to cause and symptoms.

The importance of early operation to prevent underdevelopment of the mandible and rampant dental caries should be stressed.

Extraction of all the teeth to prevent toothaches and dental infection is not recommended, as the surgical treatment of ankylosis makes good dental care possible, and because the occlusion of the teeth plays an important role in the coordination of mandibular movement after the operation. For this reason the teeth should be preserved.

Condylectomy and osteoarthrotomy are recommended rather than arthroplasty. The results of these procedures in this clinic have been very satisfactory, as they also have in the cases reported by Dingman.*

*Dingman, R. O.: Bilateral Ankylosis of the Temporomandibular Joints With Retrusion Deformity: Report of Case, J. Oral Surg. 2: 71. 1944.

HEREDITY AND CYST FORMATION

KURT H. THOMA, D.M.D., AND FRED R. BLUMENTHAL, D.M.D.

THE heredity of abnormalities of the tooth system has been studied by Keeler (1935),* who reported the occurrence of brown, transparent, fused, and impacted teeth, besides such jaw deformities as prognathism and hairlip occuring in members of several generations of families. These characteristics are passed to successive generations by genes, units of inheritance which are found in exceedingly small, filamentous structures known as chromosomes in the nuclei of either the male or female cells from which the individual arises.

That several members of a family develop cysts has probably been observed more frequently than the literature indicates. The formation of a globulomaxillary cyst in a mother, and later in her daughter has been reported (Thoma, 1944).† The rarity of this type of cyst should preclude that this occurrence was a coincidence. It has also been noted that some individuals have a constitutional disposition to cyst formation, several cysts frequently being found in one person in different parts of the upper and lower jaws. Less common is the formation of cysts in succession. One of us (Thoma) had a patient with a series of cysts which arose one after another. He was first seen in 1938 when he had a multilocular cyst in the mandible which extended from the left second incisor to the first premolar; in 1940 he had a follicular cyst in the posterior part of the right maxilla extending from the right second premolar to the second molar; in 1941 a multilocular cyst was excised from the left maxilla, which extended from the second incisor to the canine; and when last seen in 1942 a small maxilloglobular cyst was seen developing between the right maxillary second incisor and canine tooth. The x-ray is extremely interesting because it shows the point of origin of this type of fissural cyst (Fig. 545).

This paper is a report of the occurrence of cysts in three generations of one family seen for orthodontic care by one of us (Blumenthal). Two of the patients were operated on in our clinic by one of us (Thoma), and some of the others at an outside hospital. One patient was treated by Dr. B. G. Anderson of Yale University, and one by Dr. J. W. Kemper in Ann Arbor, Michigan. It is interesting to note that the cysts in this family tend to be multiple, denoting a very strong hereditary character. The family pedigree is shown in Fig. 546.

In the first generation, the grandfather had excellent teeth but developed a cyst in the lower left canine region, which was removed at the age of 78 years by Dr. O'Sullivan of Fall River; the grandmother had very poor teeth. Little else is known about their dental history. In the second generation, there were two sons and three daughters, one son and two daughters had cysts, and the

*Keeler, C. E.: Heredity in Dentistry, Dental Cosmos 77: 1147, 1935.
†Thoma, K. H.: Oral Pathology, ed. 2, St. Louis, 1944, The C. V. Mosby Co., p. 873.

remaining son and daughter were heterozygotes, the dental characteristic being recessive and transmitted to their children who developed cysts. One son had three children, the dental condition of whom is not known; the other son had three children of whom one daughter had three cysts. One daughter had two children, one with three cysts; the other was not investigated. Another daughter had five children, two with cysts. The third daughter was operated on by one



Fig. 545.—Very early formation of maxilloglobular cyst.

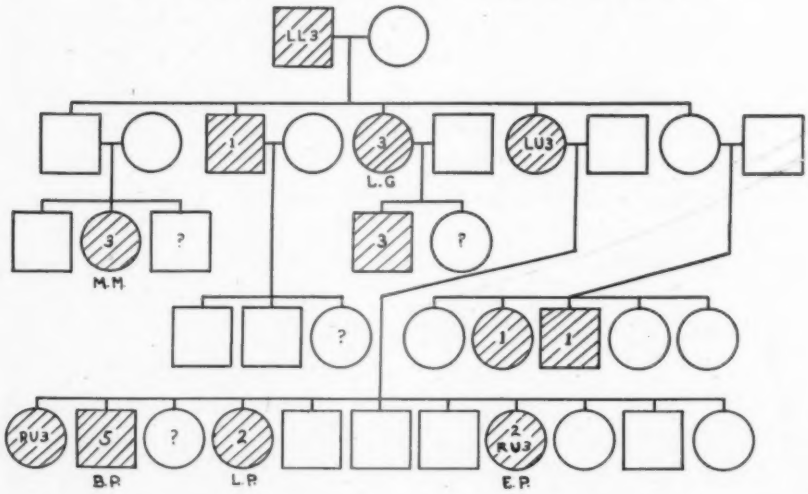


Fig. 546.—Pedigree of family with cysts. Squares indicate males; circles, females; and hatched portions, members affected with cysts.

of us (Thoma thirteen years ago for an infected dentigerous cyst connected with an unerupted left canine in the upper jaw. She had eleven children, five boys and six girls. We were fortunate to be able to make an investigation of them with the kind cooperation of Dr. J. F. Boyd, roentgenologist in Providence, R. I. One member of the family was not available as she was living in another part of the country, but of the ten children, cysts were found in four, one son and three daughters. It is interesting to note that the mother had a dentigerous cyst

formed from the left maxillary canine, while three of her children, one boy and two girls, had similar cysts, all formed from the same tooth on the other side, the right maxillary canine.

Unfortunately we were not able to get x-rays of all members of this family, therefore, only case reports of the patients we treated are included.

Case 116

L. G. (469197), a 44-year-old woman, complained of a sinus discharging into the mouth. She had had all her teeth extracted eight years ago, and has been wearing a denture since. She had had a cyst in the mandible for several years, but it gave no trouble until three years ago. Since then there has been a daily discharge from a sinus communicating with the cyst. She had had a similar cyst removed, but otherwise had been in good health.

X-ray examination showed a multilocular cyst in the ramus of the left mandible (Fig. 547), and a multilocular dentigerous cyst formed from the third molar in the right mandible (Fig. 548). In addition, there was a cyst in the left maxilla.

On Oct. 30, 1944, under gas, oxygen, and ether anesthesia, the oral cavity was prepared for operation in the usual manner. The cyst in the maxilla was operated on first. An incision was made in the left buccoalveolar sulcus, and a mucoperiosteal flap prepared and retracted. Some of the cystic fluid escaped and the cyst membrane came into view. It was detached from the bone cavity and removed. A thin, bony partition, separating the cyst from the maxillary sinus, was excised, after which a window was made in the nasoantral wall by means of an antral rasp. A dressing was inserted into the sinus with the end protruding through the nostril. The incision was closed with sutures.

The cyst in the right ramus was excised next. The incision was made along the anterior border of the ascending ramus, and extended along the alveolar crest. The mucoperiosteum was detached from the bone, and after the very thin bony wall of the cyst was removed the cyst sac could be detached. The tooth was loosened with elevators and removed with the cyst attached to it. Sulfanilamide powder was dusted into the cyst cavity, and a boric strip inserted. It was felt that this operation greatly weakened the jaw, so the other cyst was left to be removed at another time.

Examination of the tissue showed a tooth with red-gray tissue attached at the neck. This represented a cyst sac which was lined with squamous epithelium.

The postoperative course was uneventful and the patient was discharged on Nov. 3, 1944. She was then seen at regular intervals for dressings until the entire cyst cavity in the mandible had been covered with granulation tissue. She was given a syringe to irrigate the wound until it was completely healed.

Case 117

M. M., a young girl, complained of pain and swelling on the left side of the face. The submaxillary lymph nodes were enlarged and tender, and had been present for about one year.

X-ray examination showed a large cystic defect on the left in the third molar region; the tooth was absent (Fig. 549). On the right a dentigerous cyst was found (Fig. 550). In addition, there were two unerupted third molars in the maxilla.

On June 7 the patient was operated on at the Brooks Hospital. Under intravenous pentothal sodium anesthesia, the oral cavity was prepared for operation in the usual manner. An incision was made on the left over the alveolar crest starting from the anterior border of the ascending ramus, and extending to the distal surface of the second molar. From here a vertical extension was made on the outer side of the jaw. It was necessary to remove some of the bone with rongeurs and chisels in order to expose the cyst completely. The sac was dissected away from the cyst cavity and removed. A boric strip was placed into the wound.

A similar incision was made on the right, and a cyst sac exposed and incised. After a great amount of thick, yellow material containing cholesterin crystals was aspirated, the

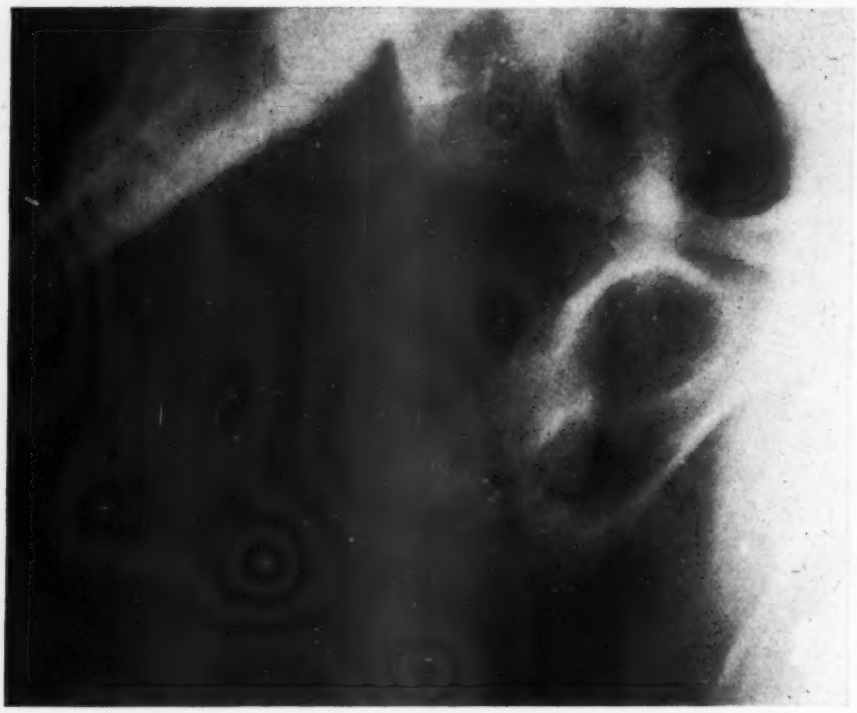


Fig. 547.



Fig. 548.

Figs. 547 and 548.—Case 116. X-rays showing multilocular dentigerous cysts, (*1*) in the left mandible, and (*2*) in the right mandible.

crown of the unerupted third molar was seen. It was loosened with elevators and removed with the cyst sac attached. The two unerupted third molars were excised next. The one on the right was found to be surrounded by a cyst sac formed from the follicle; on the left the follicle was of normal size.

The patient was allowed to go home on the sixth postoperative day, and was followed for dressings until the wounds had healed.

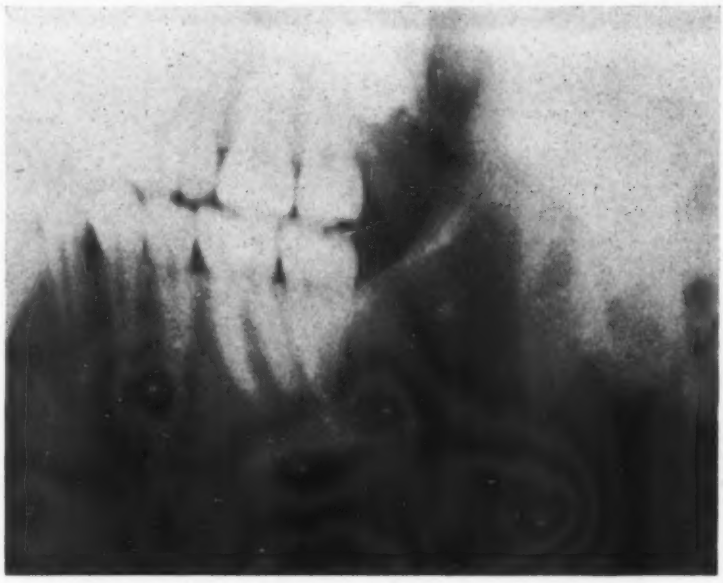


Fig. 549.



Fig. 550.

Figs. 549 and 550.—Case 117. Follicular cyst (*1*) in the left mandible, and dentigerous cyst (*2*) in the right mandible.

Case 118

B. P. was seen recently, and stated that he had been operated on four times for the removal of cysts. The first was a dentigerous cyst of the upper right maxilla which formed from an unerupted canine (*C*, Fig. 551). This cyst was typical of those inherited by other members of the family. The second cyst occurred in the lower left third molar region. This also was a dentigerous cyst containing a third molar tooth. The third was a dentigerous cyst in the lower right third molar area. The fourth cyst was discovered when a swelling developed on the upper jaw. The patient had a scar in this area. The location of the cyst and the fact that it did not contain a tooth would lead one to believe that it was a globulomaxillary cyst. The two premolars, canine, and incisors were in normal position in this segment of the jaw. The fifth cyst was in the left maxilla in the region of the third molar tooth, which had never formed. This cyst had been removed, but a few days ago a swelling appeared at the tuberosity followed by a discharge into the mouth, which brought the patient to the office for examination. A fistula was found on the posterior surface of the tuberosity into which a probe could be inserted for about ¾ inch. An x-ray taken with the probe inserted showed the presence of a residual cyst cavity. The patient was given an admission for excision.

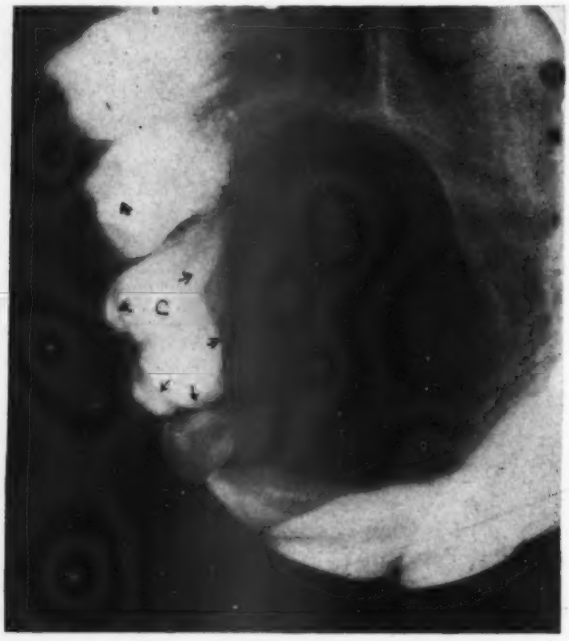


Fig. 551.—Case 118. Dentigerous cyst of maxilla with unerupted canine, *C*.

Case 119

L. P., a young woman, complained of a purulent discharge from the right third molar region in the upper jaw. Examination showed a fistula discharging some pus. A probe could be inserted for a considerable distance, and a hard, smooth object could be felt.

X-rays taken from Water's position (Fig. 552) showed an unerupted, partly formed third molar, with a radiopaque antrum. In the lateral view (Fig. 553) the tooth was seen below the floor of the orbit. There was some evidence of a narrow cystic tract. A lateral jaw film (Fig. 554) showed a second cyst of large dimensions in the ramus of the jaw.

The operation was performed at the Brooks Hospital, July 17, under avertin and ether anesthesia. An incision was made high in the buccoalveolar sulcus. The mucosa was stripped from the bone behind the zygomatic process of the maxilla, and removed by means of a chisel. The cyst came into view and the tooth could be felt. It was easily detached from

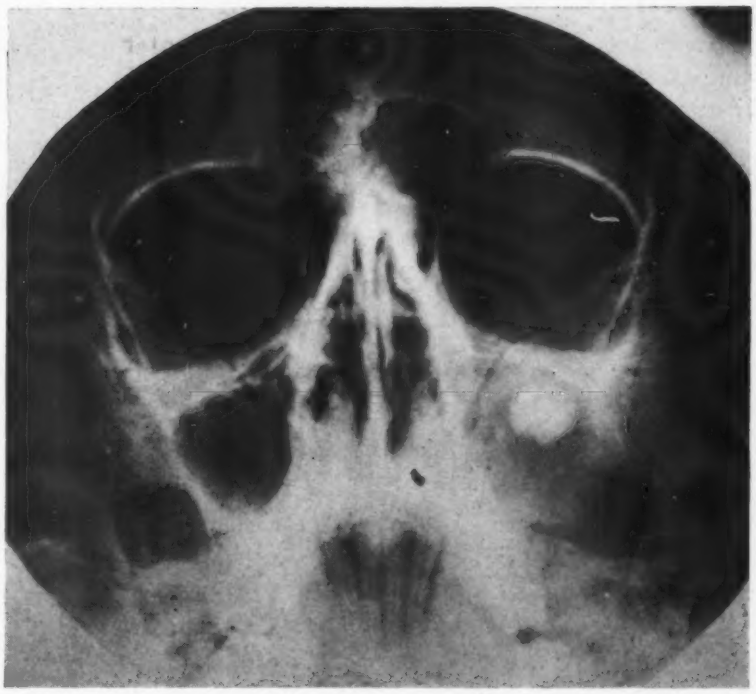


Fig. 552.—Case 119. Dentigerous cyst in maxilla in posteroanterior view.

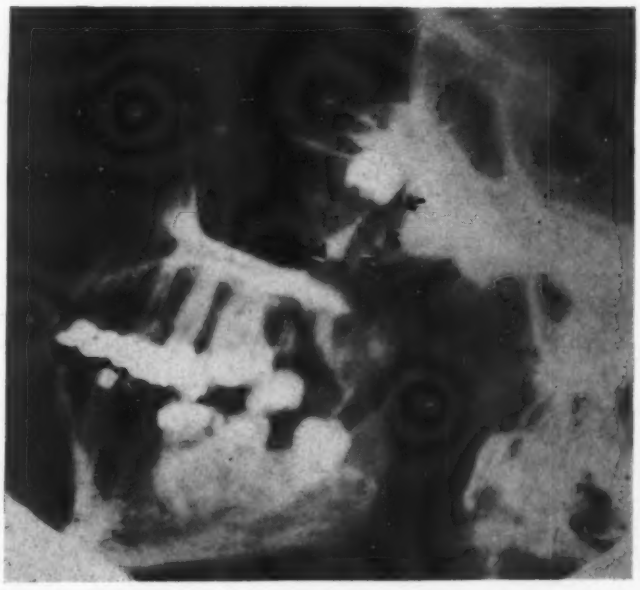


Fig. 553.—Case 119. Dentigerous cyst, lateral view.

its location and removed with the cyst sac attached. The wound was packed with a gauze strip saturated with petrolatum.

The mandibular cyst was excised next through an incision made along the anterior border of the ramus, retracting the soft tissue to each side. The cyst sac could be easily removed.

The patient was discharged from the hospital after five days, and was seen at the office for postoperative care.

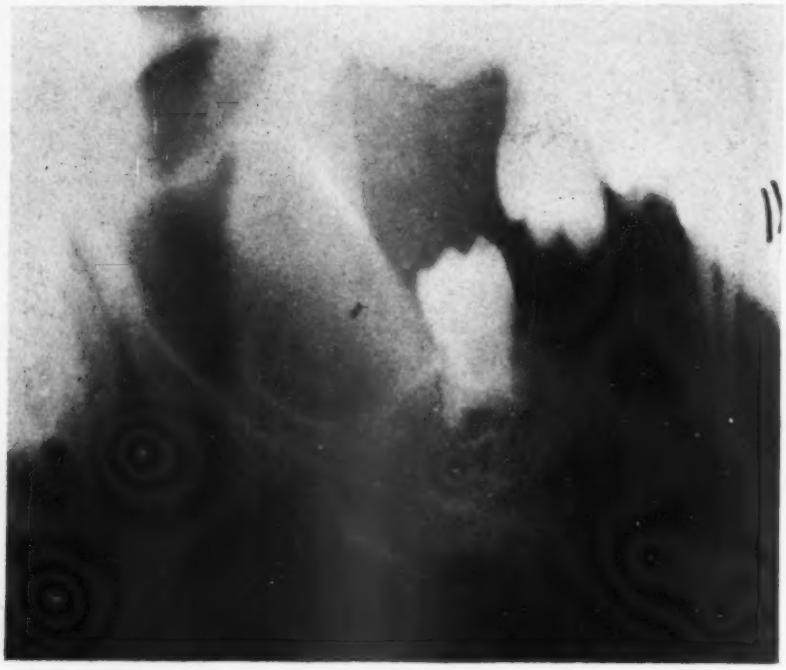


Fig. 554.—Case 119. Follicular cyst in ramus.

Case 120

E. P., a young girl, was seen on April 4 for a general x-ray examination of her teeth. The right maxillary canine tooth had never erupted. X-rays revealed a large dentigerous cyst (Fig. 555) in the upper jaw formed from an unerupted canine. The second incisor and first premolar had been somewhat displaced.

On April 5 at the Brooks Hospital, under gas, oxygen, and ether anesthesia, the oral tissues were prepared in the usual manner. A curved incision was made along the gingival margin of the palate. The mucoperiosteum was detached from the thin bony wall of the cyst, and after the bone was removed with rongeur forceps, the cyst sac was exposed and detached from the cyst cavity. It ruptured, and a clear, yellow fluid escaped. When the cyst sac was opened, this brought into view the crown of the unerupted tooth which could be loosened with elevators. The tooth was removed with the cyst sac intact. The mucoperiosteum was pressed into the bottom of the cavity and held there by means of a boric strip. A wire was drawn over it to hold the strip in place, and the ends were anchored on each side by winding them around a tooth through the interdental space. The patient was discharged from the hospital after four days.

Postoperative treatment consisted of changing the dressing and irrigating the wound with saline solution. After six days the entire bone cavity was covered by tissue. The patient was then given a syringe to irrigate the wound after meals.

She was seen again two years later when a new cyst had developed in the right mandible. There was no clinical evidence except a displacement of the two adjoining teeth, the second

incisor and canine. The x-ray showed an ovoid-shaped cystic area between the canine and second incisor causing a divergence of the roots, and extending from the alveolar process into the body of the mandible (Fig. 556). Excision was advised.



Fig. 555.

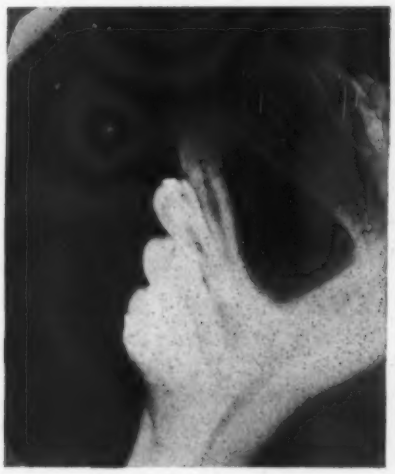


Fig. 556.

Fig. 555.—Case 120. Dentigerous cyst in maxilla.
Fig. 556.—Case 120. Follicular cyst in mandible.

On July 3, under gas, oxygen, and ether anesthesia at the Brooks Hospital, an inverted U-shaped incision was made on the outer surface of the mandible over the cyst. The mucosa was dissected away from the bone, and by means of a chisel, the outer cortex which was quite solid was chiseled away to expose the cyst sac. This was grasped with Alyce forceps, and after it was detached from its bony wall, it was removed. The cyst cavity was examined and no evidence of tooth involvement could be found. The mucosa was placed into the bottom of the cavity, and a boric strip inserted.

The patient was discharged from the hospital after three days. The postoperative treatment consisted of changing the pack and irrigating the wound. After one week the patient was given a syringe to keep the wound free of food and débris while it healed.

This cyst must have formed from an epithelial cell rest between the two teeth, and therefore should be classified a paradontal cyst.

MISCELLANEOUS CASE REPORTS

KURT H. THOMA, D.M.D., HOWARD W. SMITH, D.M.D., HAROLD F. BOSCO, D.M.D., AND HENRY M. GOLDMAN, D.M.D.

THE following cases, treated during the last six months, were thought to be of sufficient interest to be included in this issue.

Case 121

Comminuted Fracture of Mandible

N. M. (494778), a 47-year-old man, entered the Emergency Ward on June 28, 1945, with bruises and lacerations sustained shortly before in a fight. The patient's upper dental plate had been broken, and there was bleeding from the gingival margins. He had lacerations on the upper lip on the left, and the lower lip on the right. The right jaw was swollen and tender, but the patient could move it fairly well. No fracture could be felt on palpation. The bruises were treated and an ice pack applied to the jaw. The patient went home, against advice, before an x-ray examination could be made.

It was not until July 2 that the patient returned to the Outpatient Department to have the x-rays taken. They revealed multiple fractures, and the patient was admitted to the House.

Examination showed a moderate-sized swelling of the right mandible, extending superiorly to the zygoma, but not localized. The patient was partly edentulous. Manipulation of the mandible did not reveal any displacement. The patient, however, was unable to open the jaws wide because of pain.

X-ray examination showed a comminuted fracture of the horizontal ramus of the right lower jaw (Fig. 557), and a subcondylar fracture with considerable displacement and overriding of the fragments.

The patient was given 96,000 units of penicillin intramuscularly each day. A Gunning splint of acrylic material was constructed to immobilize the mandible.

On July 6 circumferential wiring of the jaw was performed under endotracheal gas, oxygen, and ether anesthesia. The face and mouth were prepared with iodine, and a long lumbar-puncture needle was inserted under the right mandible so that it emerged on the inner aspect. A 22 gauge stainless steel wire was inserted into the needle and drawn into the mouth. The needle was removed, leaving the wire in place. The needle was again inserted, this time in the mouth along the buccal side of the bone, emerging at the same puncture in the skin. The end of the wire was inserted into the needle, and the needle removed, leaving the wire in place. The prepared splint was inserted and the wire twisted over it so as to hold the comminuted fracture immobilized (Fig. 558). The upper splint was then inserted and the mandible placed in centric occlusion. A traction bandage was applied.

The penicillin was continued for four days postoperatively. The patient was given a high-calorie liquid diet. The hemoglobin was 11.6 Gm., and the white cell count 9,450.

Postoperative x-rays showed that the fragments were held in good position by the wire (Fig. 558). The displacement of the fragment of the coronoid process was not much changed.

The patient made an uneventful recovery and was discharged on July 11 to be followed in the Outpatient Department. He returned twice weekly to have the bandage changed and the mouth sprayed out. An x-ray on August 7 showed that good position of the fragments was maintained, but no definite callus could be seen. On August 10 the bandage was removed. On August 17 the circumferential wire was removed, and on August 31 the patient was discharged relieved.

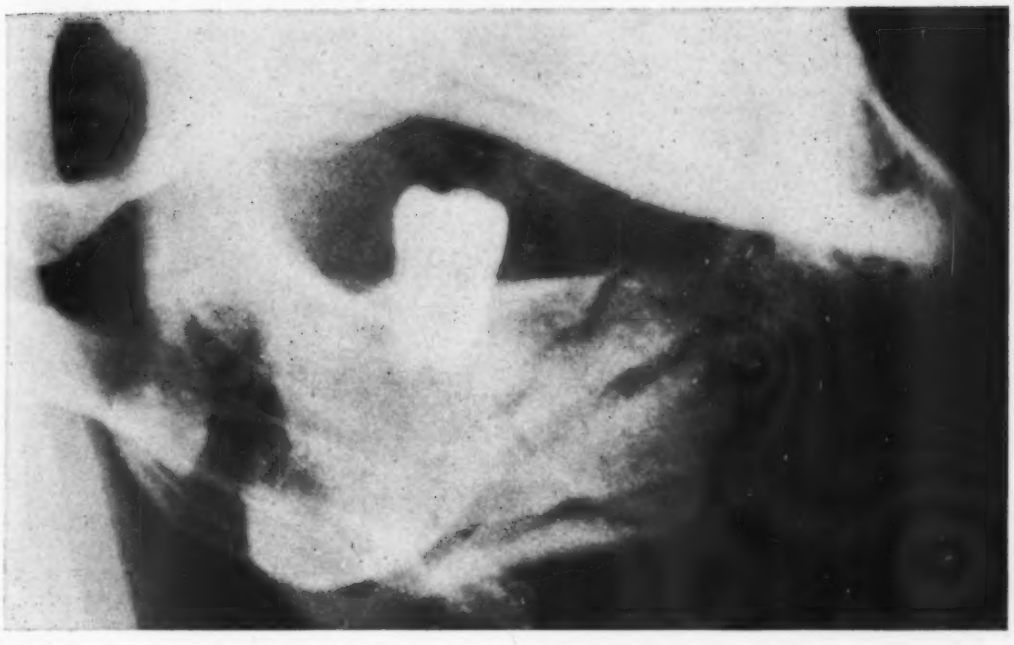


Fig. 557.—Case 121. Comminuted fracture of the mandible.

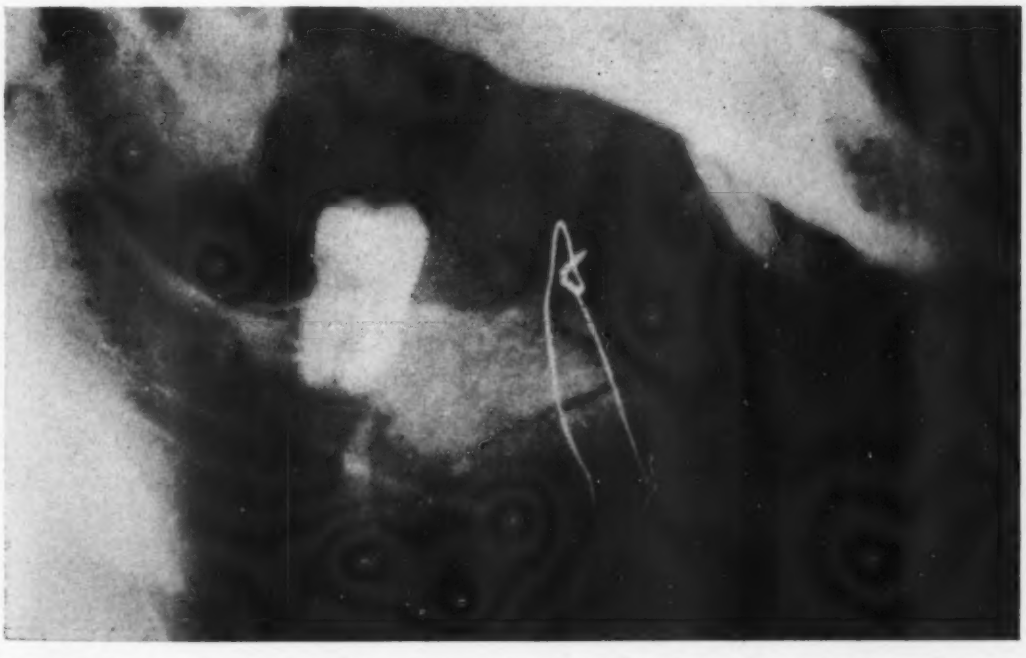


Fig. 558.—Case 121. Comminuted fracture of the mandible reduced and immobilized by a circumferential wire around an acrylic splint.

Case 122

Compound Fracture of Mandible With Displacement of the Posterior Fragment

M. P. (479286), a 45-year-old woman, came to the dental clinic on Jan. 11, 1946, complaining of a fractured jaw.

Six days before, during a fight, the patient had received multiple bruises and an injury to her jaw, which she treated with cold packs. There was no improvement of the jaw, however, so she went to her dentist who referred her to the hospital.

Examination showed a well-developed, somewhat obese woman with blackened eyes, a hemorrhagic left conjunctiva and sclera, two small ecchymotic spots on the right thorax, and a swelling of the lower right jaw. The jaws could not be opened more than 2 cm.

X-ray examination of the jaws and facial bones showed a fracture of the right lower jaw through the socket of the remaining molar; there was considerable displacement (Fig. 559). There was no evidence of fracture of the zygoma or other facial bones.

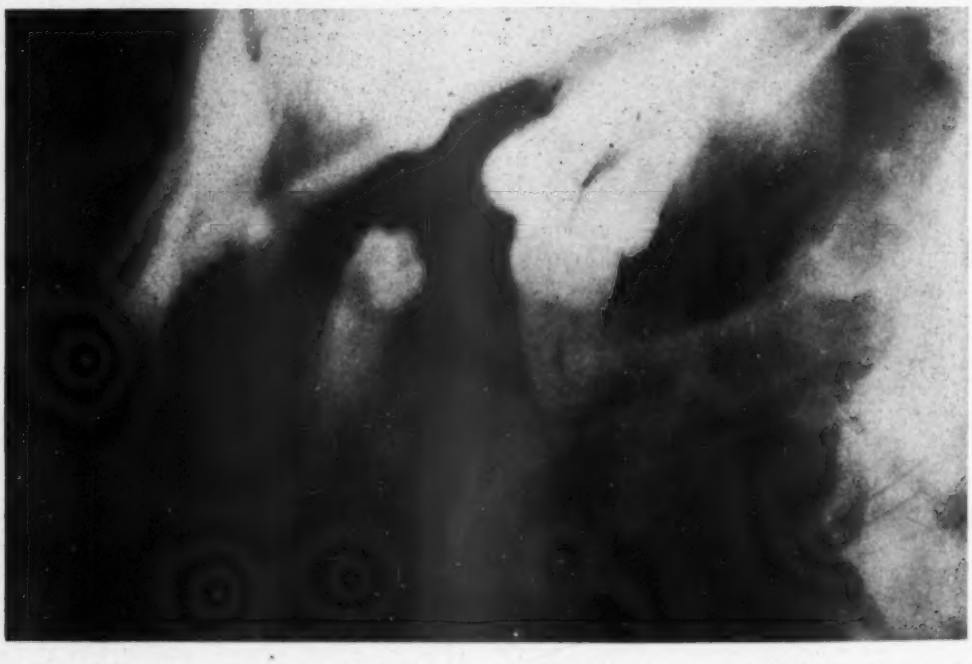


Fig. 559.—Case 122. Fracture of the mandible compounded into the mouth with displacement.

Diagnosis: Fracture of the lower right mandible and multiple bruises.

The patient was admitted to the hospital on January 11, and put on a high-calorie, high-vitamin liquid diet. The fracture was temporarily immobilized with a Barton bandage which held the anterior fragment in position. Blood studies showed a white cell count of 12,500, hemoglobin 14.5 Gm., nonprotein nitrogen 15 mg. per liter, protein 7.7 Gm. per liter, Van den Bergh's test $\frac{D}{I} = \frac{0.4}{1.1}$, and prothrombin time 25 seconds.

Three days after admission the left eye was clearing gradually and the right submaxillary swelling was less pronounced. On January 15 under endotracheal gas, oxygen, and ether anesthesia, an open reduction was performed. After the usual preparation of the face and mouth, a skin incision was made parallel to the inferior border of the mandible and about ¾ inch below. The platysma was divided. The external maxillary artery was bisected, and the periosteum incised over the fracture site. The anterior and posterior fragments were exposed, and holes were drilled, one on each side of the fracture line about 0.3 cm. from the inferior border and about 1 cm. from the fracture line. A 24 gauge stainless steel wire was

inserted and tightened, bringing about a hairline reduction of the fracture. The periosteum was closed; the subcutaneous tissues were sutured with catgut and the skin with interrupted dermalon sutures. A dry sterile dressing was applied.

Next, the mouth was opened and the throat packed. The lower right molar which was

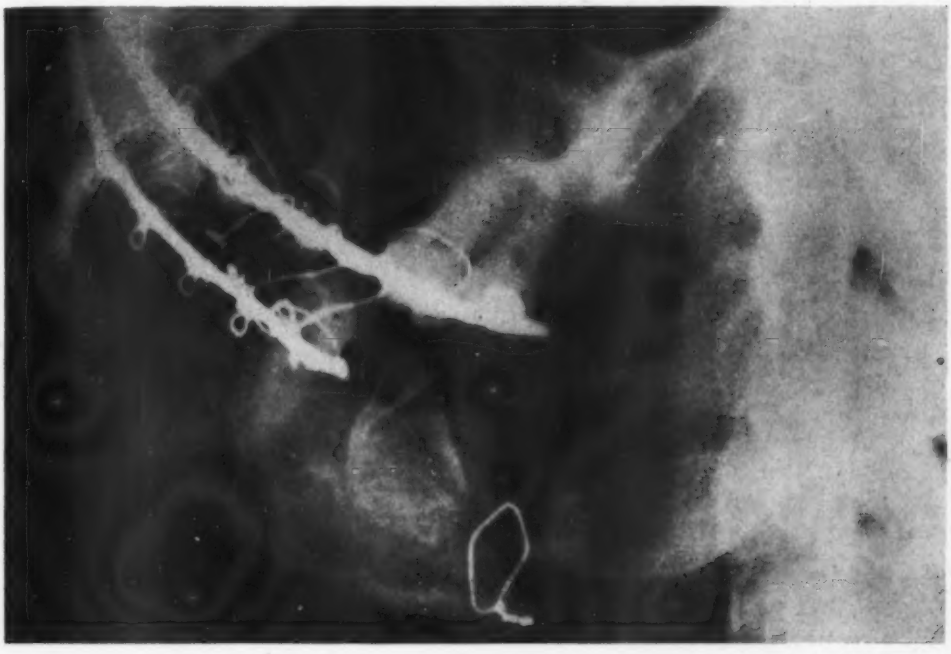


Fig. 560.—Case 122. Fracture ineffectively fixed by means of horizontal wire which allowed the fracture to be displaced while the patient was recovering from the anesthesia.

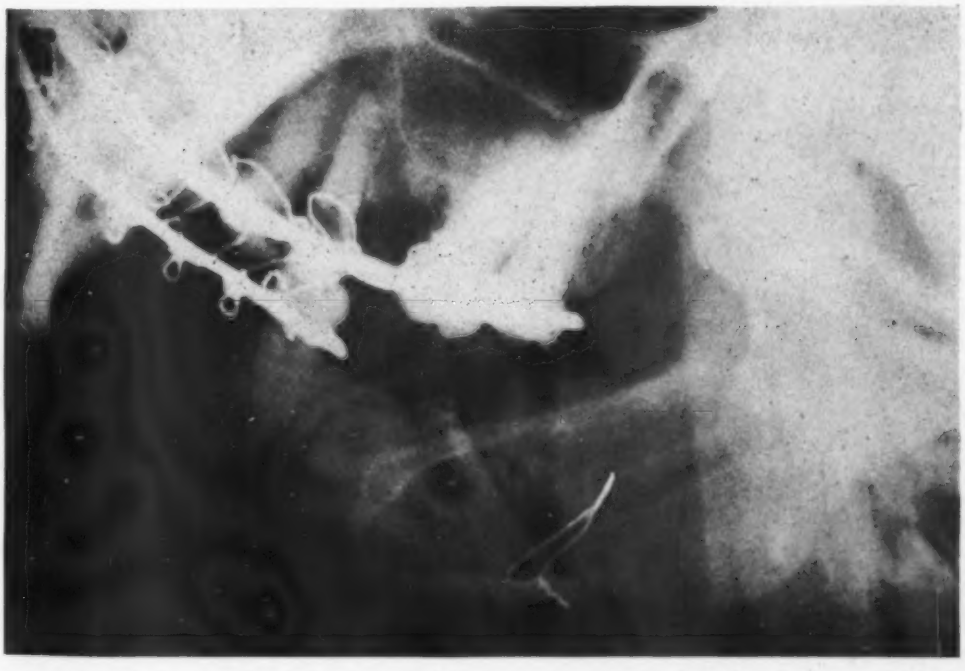


Fig. 561.—Case 122. X-ray showing revision of reduction by means of an oblique transosseous wire.

in the fracture line was removed, and also the lower right central incisor and a root in the premolar region. Jelenko splints were applied to the upper and lower arches, and the mandible immobilized by the use of rubber bands. The second day after the operation the rubber bands were replaced with four 25 gauge stainless steel wires. A pressure bandage was applied to the external wound surface.

The patient was given an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in saline and 16,000 units of penicillin intramuscularly after the operation. On the second postoperative day the patient's temperature rose to 102° F. It was believed that solid material had been aspirated during intubation; therefore, she was given penicillin, 16,000 units intramuscularly every three hours, and sulfadiazine, 2 Gm. to start and 2 Gm. every four hours. The sulfadiazine was discontinued on the fifth postoperative day.

On January 17 the teeth were in good position, but the x-ray examination showed that the fracture was displaced (Fig. 560). The transosseous wire had failed to keep the fragments impacted. On January 20, under endotracheal gas, oxygen, and ether anesthesia, a revision of the reduction was performed. After the usual preparation of the face and mouth, the skin sutures were removed from the previous operative site. The fracture was exposed and it was found that there had been an anterior, medial, and superior shifting of the posterior fragments, without a dislodgment of the wires from the fragments; the wire was still in the two previously made holes. The fracture had shifted, probably when the patient was coming out of the ether after the first operation. A new hole was drilled in the posterior fragment above the mandibular canal, and about ⅜ inch posterior to the fracture line. A 24 gauge stainless steel wire was passed through this hole and the former hole in the anterior fragment, drawing the posterior fragment down into position. The periosteum was closed with catgut, and the skin with a subcuticular suture. The intermaxillary wires which were cut prior to the operation were replaced. The patient received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose and saline after the operation.

An x-ray examination two days later showed that now there was close approximation of the fragments (Fig. 561). The fracture line was a narrow linear area of decreased density.

The patient made an uneventful recovery, and was discharged on January 26 to be followed in the Outpatient Department.

Discussion.—This case demonstrates that in oblique fractures the wiring must be of such a nature that disimpaction is prevented. In the first instance the fragments could slide over each other, slacking the wire which made it possible for the posterior fragment to displace upward. The second wire was placed to prevent this.

Case 123

Compound Fracture of the Right Mandible and Subcondylar Fracture on the Left

D. O'B. (495145), a 52-year-old man, came to the Emergency Ward on July 2, 1945, complaining of inability to open the mouth and pain in the right mandible. The day before, while going upstairs, he fell and struck the right side of the face on a step.

Examination showed marked asymmetry of the face due to a moderate soft-tissue swelling in the right mandibular area, extending from the angle of the jaw to the corner of the mouth, and about 1 inch below the lower border of the mandible. Testing showed that there was motion in the mandible posterior to the right canine, and there was elevation of this fragment.

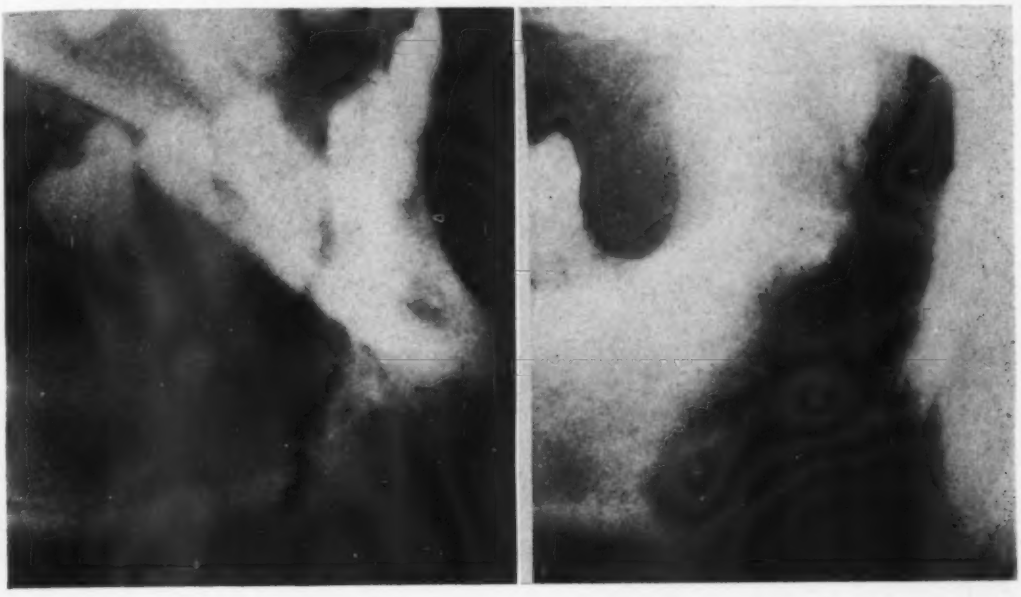
X-ray examination showed a fracture of the right lower jaw through the socket of the first premolar with a slight displacement of the posterior fragment (Fig. 562). There also was a fracture through the base of the left condyloid process without any appreciable displacement (Fig. 563).

Diagnosis: Compound fracture of the right mandible and left subcondylar fracture on the left.

On July 3 under local anesthesia Jelenko splints were applied. The first premolar in

the line of fracture was extracted and also the canine which was loosened by the accident. An Ace bandage for temporary immobilization was applied.

The patient was admitted to the House on July 4. The white blood count on July 2 was 7,900, and on July 4, it was 13,000; the hemoglobin was 13 Gm. He was given penicillin, 48,000 units intramuscularly on the first hospital day, and 96,000 units on each of the next six days. A plaster head cap was also made.



Figs. 562 and 563.—Case 123. Double mandibular fracture, one in the mental region on the right, and one in the subcondylar region on the left.

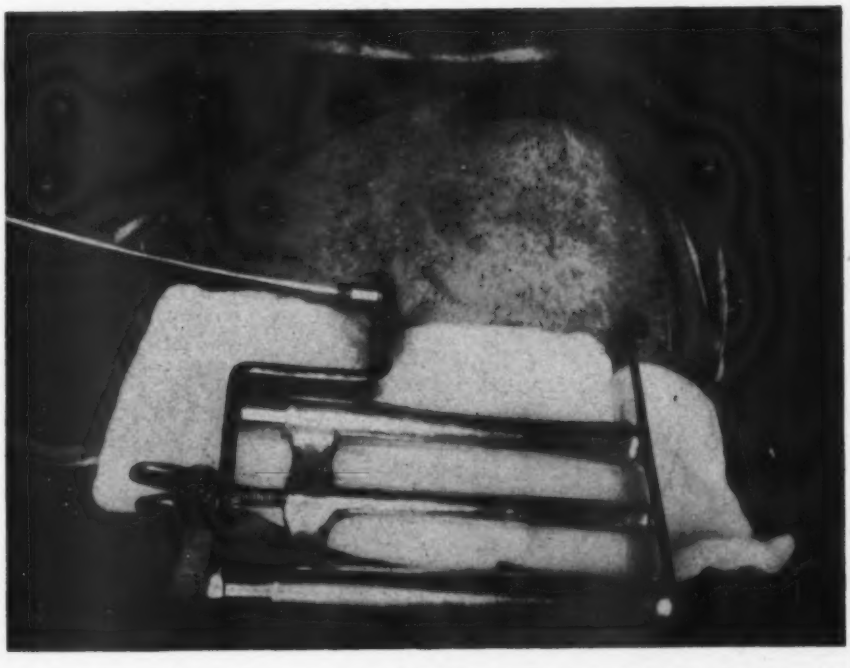


Fig. 564.—Case 123. Transfixation of the anterior part of the mandible by means of a Kirschner wire, showing the use of the Pincock pin guide.



Fig. 565.—Case 123. Photograph showing cranial mandibular fixation by means of a Kirschner wire through the anterior part of the mandible and Frac-Sure rods.

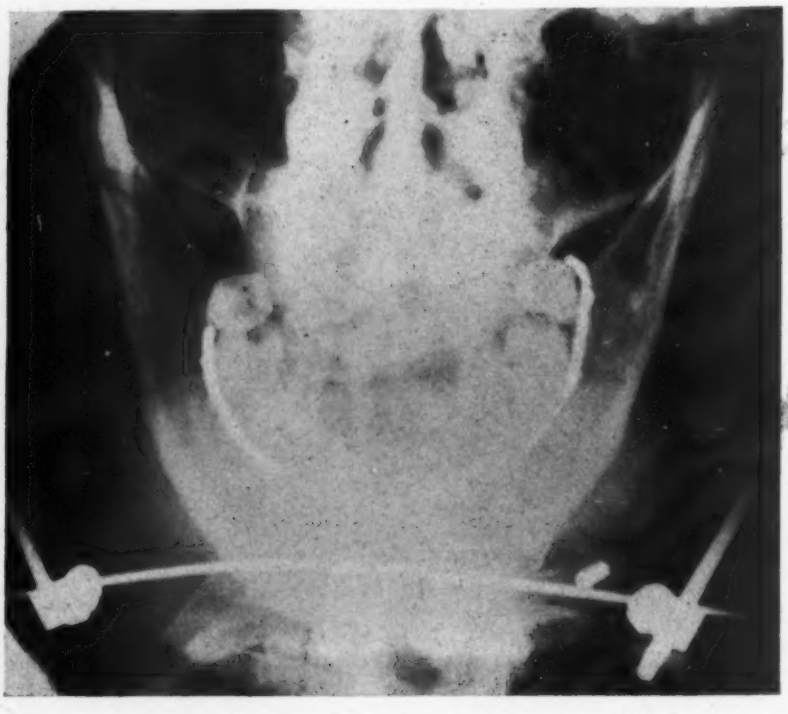


Fig. 566.—Case 123. Postreduction x-ray.

On July 6 under intratracheal gas, oxygen, and ether anesthesia, an open reduction of the fracture of the right mandible was performed. A 22 gauge stainless steel wire was used for transosseous fixation. The incision was closed with Dermalon sutures. A Steinmann pin was inserted horizontally through the anterior portion of the mandible (Fig. 564), and Frac-Sure appliances were attached to this and connected with the rods on the plaster head cap (Fig. 565).

X-ray examination on July 9 showed the Steinmann pin through the mandible; the fragments were in good position (Fig. 566).

The postoperative recovery was uneventful and the patient was discharged on July 12 to be followed in the Outpatient Department. He was seen periodically to have the dressings changed. There was no infection around the Steinmann pin. On August 15 the head cap and Steinmann pins were removed. One week later the Jelenko splints were taken off, and the fracture seemed to be firm.

Case 124

Submaxillary Abscess Due to Acute Pericoronitis Treated by Aspiration and Instillation of Penicillin

J. G. (12837), a 38-year-old man, was admitted to the emergency ward on Dec. 19, 1945, with a swelling of the left jaw and pain.

The patient stated that the jaw had been swollen for the past five days and that he had had a temperature of from 100° to 102° F. accompanied by chills and night sweats. The pain had gradually increased in intensity and was worse at night. He had had no local treatment.

Examination revealed a large semifluctuant mass above the angle of the left jaw. There was trismus and the patient could open the mouth only about 1 cm. The breath was foul. Intraorally there was visible a partially erupted lower third molar which was impacted against the second molar. Pus could be seen draining from the inflamed gingival tissues surrounding the third molar. The white cell count was 24,500, and the temperature 101.8° F.

X-ray examination showed the bone of the left side of the lower jaw to be normal in texture and density. There were areas of rarefaction about the root of the second premolar which was root-filled, and about the anterior root of the second molar. The first molar was carious. The third molar was impacted with bone destruction below the mesial side. There was no evidence of osteomyelitis (Fig. 567).

Diagnosis: Acute pericoronitis about the partially erupted third molar, chronic apical infection of the second premolar and second molar.

On December 21 the patient was admitted to the House. He was given penicillin, 24,000 units intramuscularly the first day, and 96,000 units on each of the next six days. Under endotracheal gas, oxygen, and ether anesthesia the lower left second premolar, the first, second, and third molars were extracted. About 30 c.c. of foul pus was aspirated from the abscess, and 6 c.c. of penicillin injected (Fig. 568). The culture of the pus showed abundant alpha hemolytic streptococci. The patient received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water. Flaxseed poultices were applied to the jaw every three hours.

On December 23, under sterile procedures, about 35 c.c. of pus were aspirated from the abscess area, and 32,000 units of penicillin injected. Flaxseed poultices were continued, and hot saline irrigations every three hours were prescribed. The temperature was normal.

On December 27 there was still some intraoral drainage of pus from the site of the wound, but no drainage extraorally. The swelling had receded, although the submaxillary area on the left was still somewhat indurated. The patient was discharged on December 28, to be followed in the Outpatient Department.

He was seen three days later when the swelling had almost disappeared. The intraoral wounds were healing well. X-ray examination showed the empty alveolar sockets in the lower jaw. No definite osteomyelitis could be seen. When he was again seen, three weeks later, the extraoral swelling had completely receded, and he was discharged.

This treatment is being carried out as an experiment in a series of cases to see if, by aspiration of the pus and instillation of penicillin into the abscess cavity combined with parenteral injections of penicillin, incision will be unnecessary.



Fig. 567.—Case 124. X-ray showing periapical infection about the roots of the second premolar and caries with pulp infection.



Fig. 568.—Case 124. Submaxillary abscess showing aspiration of pus with a Luer-Lok syringe.

Case 125

Osteomyelitis of the Mandible

R. W. (507899), a 46-year-old man, was admitted to the hospital on Oct. 17, 1945, because of chronic osteomyelitis of the mandible, for saucerization and sequestrectomy.



Fig. 569.—Case 125. X-ray showing a small erosion in the bone at the site of a chronic fistula.

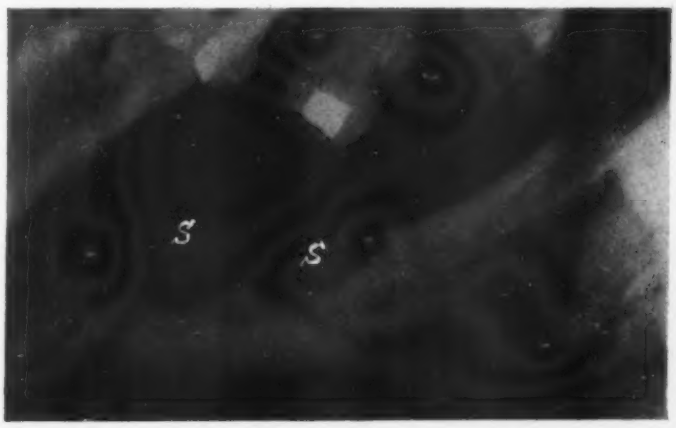


Fig. 570.—Case 125. X-ray showing large sequestra (*S, S*).

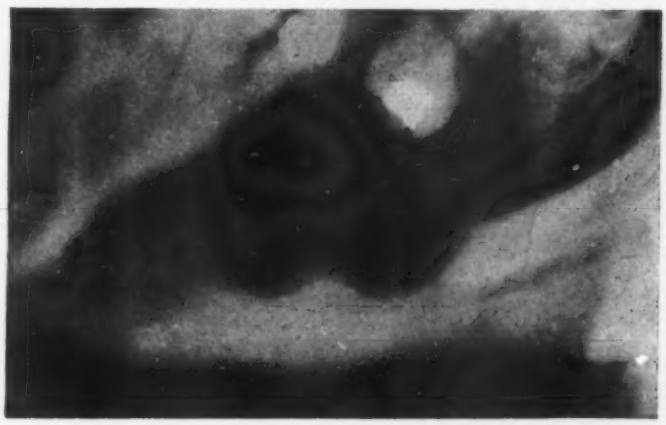


Fig. 571.—Case 125. Postoperative x-ray after sequestrectomy.

Ten years before the lower left molars had been extracted. For four months prior to entry, the patient had been treated for chronic osteomyelitis of the left mandible. An x-ray taken at that time showed a small erosion at the site of a chronic fistula (Fig. 569).

When the patient was admitted to the hospital, intraoral examination showed a nontender swelling of the left maxilla. There was a scar under the chin. Intraorally there was a fistula on the lower left alveolar ridge draining a small amount of mucopurulent sanguineous material. There were two hard submaxillary nodes palpable on this side.

X-ray examination revealed a long irregular section of the alveolar process detached from the underlying bone (Fig. 570). It was more highly calcified than the main part of the mandible. Diagnosis: chronic osteomyelitis with sequestration.

On October 18 under endotracheal gas, oxygen, and ether anesthesia, the mouth was prepared with zephiran and an incision was made over the alveolar crest of the left mandible from the ascending ramus to the canine tooth. The mucoperiosteum was reflected, and what looked like a sequestrum in the x-ray was found to be firmly attached to the jaw. The bone, however, looked very necrotic. It was separated from the normal bone by means of osteotomes and removed. All sharp edges of the bone were made smooth. There was considerable bleeding, which was arrested with fibrin foam and thrombin. The mucosa was drawn over the osteotomized bone, and closed with dermalon sutures.

The patient received 108,000 units of penicillin intramuscularly on the day of operation, and also on the next day. A culture from the discharge showed moderate alpha hemolytic streptococci, a few *Staphylococcus aureus*, and rare *Staphylococcus albus*.

Postoperative x-ray examination showed the sequestrum removed (Fig. 571). Recovery was uneventful. The patient was discharged on the second postoperative day, and given oral penicillin* which he took four times a day on an empty stomach, 50,000 units each time. The sutures were removed on the eighth postoperative day. The wound healed by first intention.

Pathologic report: The specimen consisted of two pieces of bone measuring 1 by 0.7 by 0.2 cm. each, and several smaller chips. Microscopic examination revealed fragmented pieces of bone containing empty lacunae. In the marrow spaces there was much debris, and some showed marked inflammatory infiltration composed of large mononuclear cells, some plasma cells, and a few lymphocytes. In other areas numerous polymorphonuclear leucocytes were seen. Diagnosis: osteomyelitis.

Case 126

Ranula

J. G. (512629), a 16-year-old boy, came to the Dental Clinic on Nov. 27, 1945, complaining of a swelling in the floor of the mouth of two months' duration.

While playing football three months before, the patient was kicked in the lower jaw in the region of the sublingual glands. There were no external lacerations, bleeding, tenderness, or pain. One month later, however, the patient noticed that a swelling had formed under the chin, and while brushing his teeth, he noted a large swelling under the tongue. He had not experienced any pain or tenderness either before or after meals. This swelling had gradually increased in size. About three weeks before, he had noted on five occasions that a fine stream of colorless, tasteless, waterlike material squirted from the area in the floor of the mouth. There was neither pain nor abnormal sensation.

Examination revealed a bluish-white, soft, nontender mass, 6 by 4 cm., in the floor of the mouth, filled with fluid. It was more marked on the right and extended over the midline (Fig. 573). Externally a swelling could be seen in the sublingual area on the right (Fig. 572); it felt fluctuant on palpation, and could be transilluminated. Diagnosis: Ranula.

On December 3 the patient was admitted to the House for excision of the ranula. The red blood count was 4,750,000, the white count, 9,050, and the hemoglobin 15.5 Gm.

*Supplied by Hoffman-LaRoche for experimental purposes.

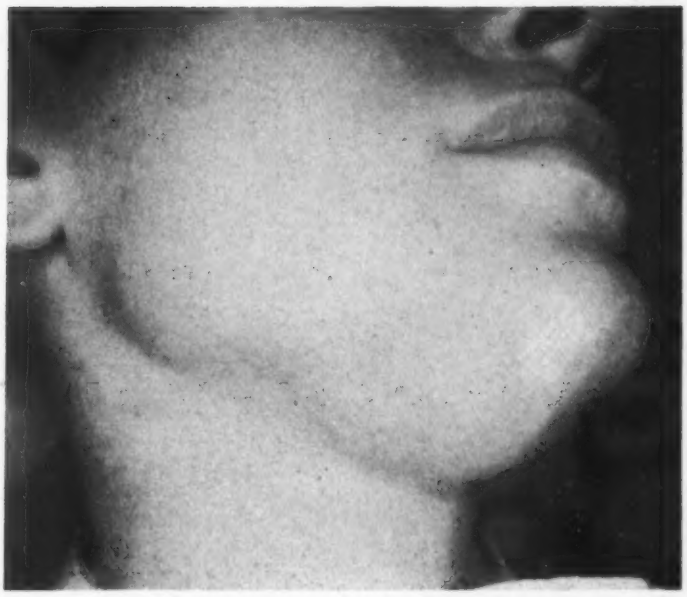


Fig. 572.—Case 126. Photograph showing submental swelling due to ranula.



Fig. 573.—Case 126. Photograph showing intraoral appearance of ranula.

The next day, excision was performed under endotracheal gas, oxygen, and ether anesthesia. The mucous membrane in the floor of the mouth overlying the cyst was infiltrated with 1 per cent monocaine and 1:75,000 epinephrine for hemostasis. The mucous membrane was incised to remove the roof over the cyst. A cyst membrane was found to lie directly under the covering of mucous membrane. This was penetrated, and about 35 c.c. of thick, yellow, translucent material was withdrawn. Within this cyst another cyst was found. The second cyst was excised, and the periphery of the first cyst was sutured to the floor of the mouth, thus providing an epithelial covering and eliminating the cyst cavity. It was sutured with five dermalon sutures (Fig. 574). Bleeding was controlled without difficulty. The patient left the operating room in good condition. He received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water during the operation.

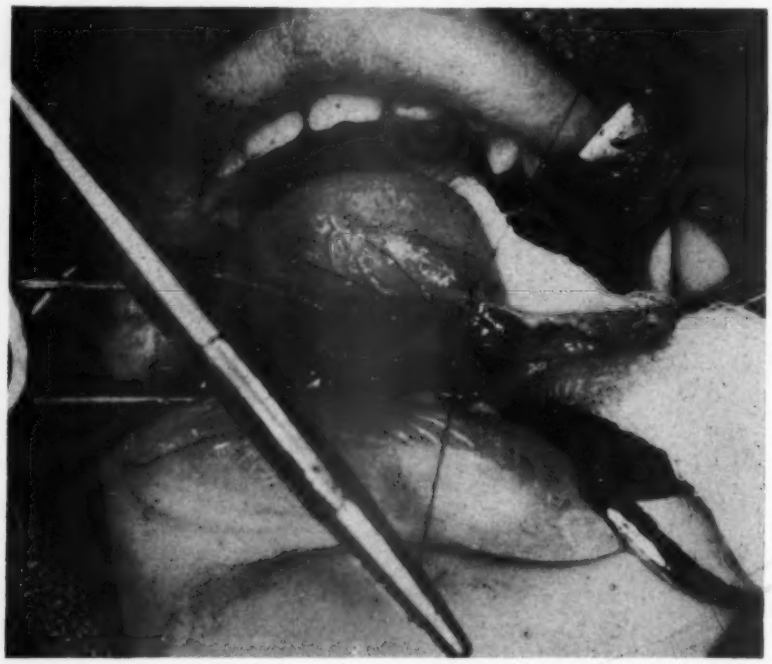


Fig. 574.—Case 126. Photograph showing excision of ranula and suture of floor of ranula to floor of mouth.

On the first postoperative day there was some edema of the floor of the mouth and swelling in the submental area. The patient had difficulty in swallowing. Hot saline irrigations were given every half hour, and an ice bag applied to the neck. On the next day the patient had less difficulty in swallowing and could open his mouth wider. He was given 60,000 units of penicillin intramuscularly, and 84,000 units on the next day. On the third postoperative day the patient could eat and drink comfortably. He was discharged on December 8 to be followed in the Outpatient Department. Recovery was uneventful.

Case 127

Dentigerous Cyst of Maxilla; Excision With Preservation of the Teeth

J. K. (512326), a 6-year-old boy, was admitted to the hospital on Nov. 27, 1945, with a diagnosis of maxillary cyst.

Two years before, the patient had fallen against a table and lacerated the gingiva in the region of the upper right canine. This healed without event. One and one-half months before, the patient's mother had noted slight asymmetry of the face, particularly over the right upper lip. There was no history of pain or drainage.

Examination showed slight asymmetry of the face over the right upper lip. Intraorally there was a swelling of the alveolar bone in the region of the right canine fossa.

X-ray examination revealed a large radiolucent area in the anterior part of the maxilla in the region where the permanent incisors should form (Fig. 575). The left first and second incisors were found to be developing normally. The right first and second incisors could be seen crowded into the canine region. The deciduous teeth showed evidence of resorption. Diagnosis: Dentigerous cyst.

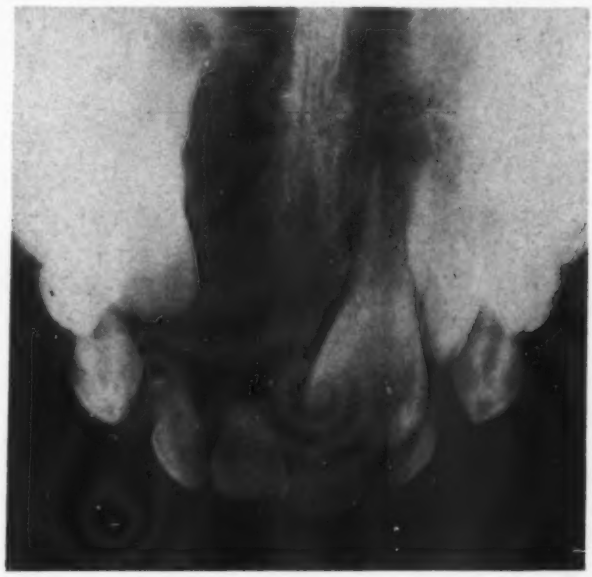


Fig. 575.—Case 127. X-ray showing a cystic area with the right upper first and second permanent incisors displaced and the roots of the deciduous teeth resorbed.

On November 28, under insufflation gas, oxygen, and ether anesthesia, a U-shaped flap was made in the labial aspect of the maxilla over the bulging prominence of the cyst. The mucoperiosteum was dissected away from the thin bony cyst wall. It was perforated at one place and thick, creamy pus exuded. A window was then cut through the thin bone, exposing the cyst. The pus was aspirated, and it was found that the crown of the permanent central incisor was protruding into the cyst. Therefore, this was a dentigerous cyst formed from the follicle of the first permanent incisor. The thin cyst membrane was detached from the bony wall, until it remained attached only at the neck of the incisor. While the incisor was held in place with an instrument, the membrane was excised from around the tooth and removed (Fig. 576). The two deciduous incisors were then extracted. The mucoperiosteal flap was placed over the crown of the permanent incisor, and a boric-strip pack was placed into the wound.

The postoperative course was uneventful. The wound was irrigated with saline solution daily, and an iodoform gauze pack impregnated with petrolatum was inserted. The patient was discharged on December 3 to be followed at the office. After eight weeks the cyst cavity was fairly well healed; the tooth had perforated the mucosa and had begun to erupt (Fig. 577).

Pathologic examination: The specimen consisted of a thin, smooth-walled, reddish cyst, measuring 1.5 cm., and a piece of bone measuring 1 by 0.5 cm. Microscopic examination revealed a strip of dense collagen connective tissue densely infiltrated by plasma cells. Numerous capillaries were present and filled with blood cells. The connective tissue was covered by stratified squamous epithelium which was thick in some areas and entirely missing in others. Diagnosis: Dentigerous cyst.

The patient was seen once a month after this so that the eruption of the teeth could be observed. On March 10 the second as well as the first incisor was erupting, although out of their normal positions (Fig. 578). The x-ray taken at this time showed the roots develop-

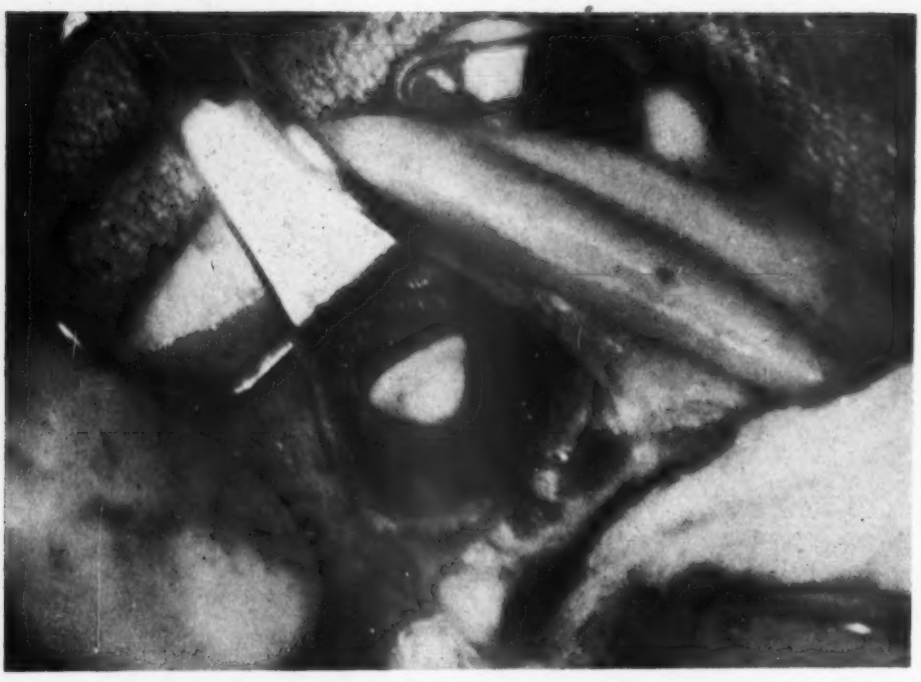


Fig. 576.—Case 127. Photograph of maxillary dentigerous cyst showing the crown of the permanent central incisor after excision of the cyst sac.

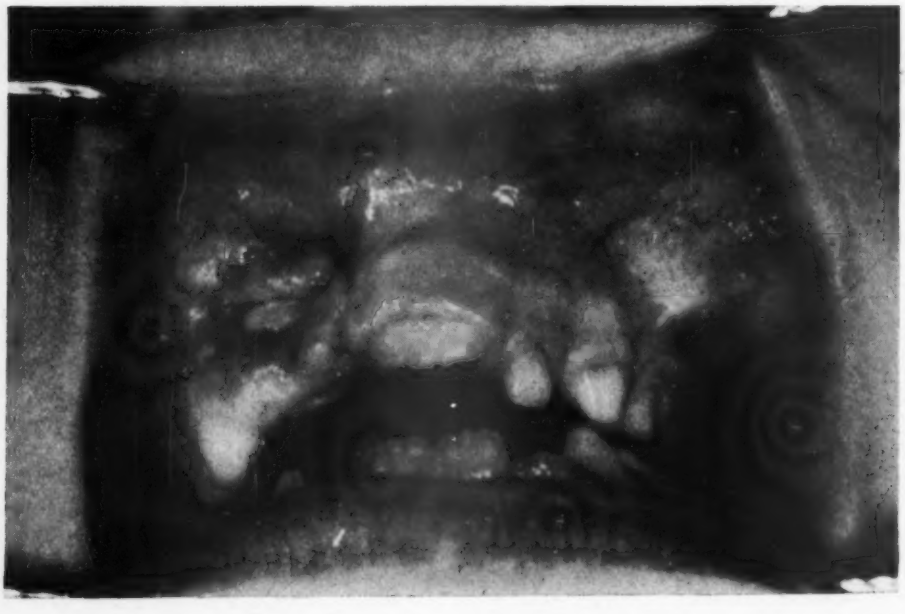


Fig. 577.—Case 127. Photograph taken eight weeks later showing eruption of the right first incisor, which was allowed to remain when the cyst was removed.

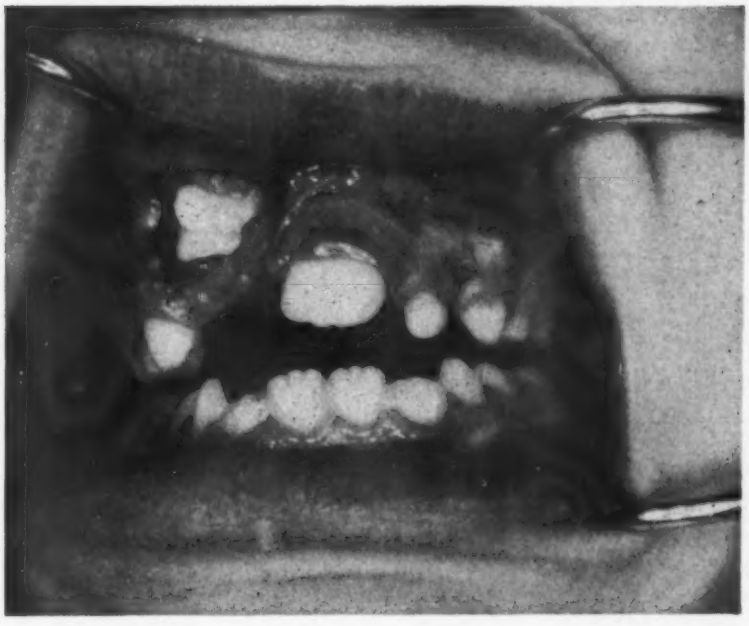


Fig. 578.—Case 127. Photograph taken three months later showing eruption of the first and second incisors.

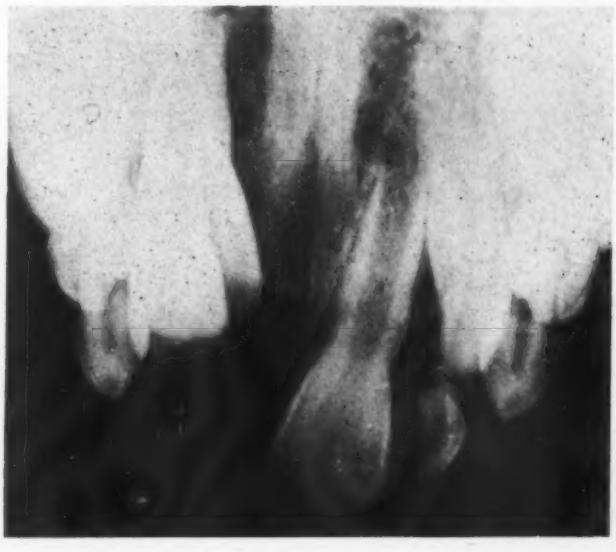


Fig. 579.—Case 127. X-ray showing the eruption of the first and second incisors.

ing normally (Fig. 579). The orthodontist who was consulted recommended that the teeth be banded in about six months and aligned in proper occlusion.

Case 128

Dentigerous Cyst of Maxilla Formed From a Mesiodens

R. W. (506632), a 13-year-old boy, came to the Tumor Clinic on Oct. 4, 1945, complaining of a swelling of the left side of the maxilla and irregularity of the dentition. The mass had been present for the past five years, and during the last year it had produced deviation of the nasal septum.



Fig. 580.—Case 128. Photograph showing deformity of the middle face due to cyst formation.

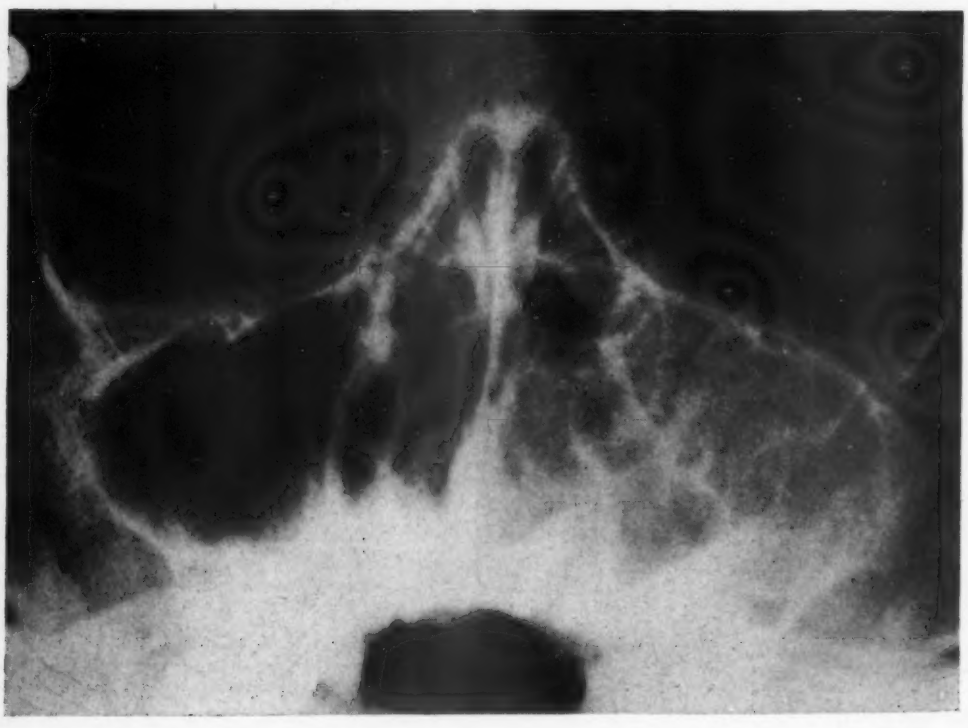


Fig. 581.—Case 128. X-ray showing large cystic mass on the left side of the maxilla displacing the nasoantral wall.

Examination showed an enlargement of the left maxilla, involving the entire left side and causing irregularity of the teeth (Fig. 580). There was a deviation of the nasal septum to the right.

X-ray examination showed a large homogenous soft tissue mass arising from the left side of the maxilla or antrum, (Fig. 581), displacing the teeth upward and outward, and extending the medial wall of the antrum into the nose. A small mesiodens was seen (Fig. 582) in the cyst. The lateral wall of the right antrum was sharply defined and showed no evidence of bone destruction. The roof of the antrum was somewhat thin compared with the other side.

Impression: Tumor, either benign giant-cell, sarcoma, or fibro-osteoma.

On October 9 the patient was seen in Tumor Conference, and the mass aspirated. A free-flowing dark brown fluid was obtained without difficulty, indicating that this was a dentigerous cyst.



Fig. 582.—Case 128. Occlusal film showing a polycystic area with a mesiodens from which the cyst had formed.

Subsequent to this examination the cyst swelled up, pained him severely, and ruptured. A great deal of bloody fluid drained away. The patient was transferred to the Dental Clinic, and admitted to the House on Nov. 5, 1945, for excision of the cyst.

On November 6 under insufflation gas, oxygen, and ether anesthesia, the cyst was excised. The mouth was prepared in the usual manner and a postnasal pack inserted. After monocaine epinephrine was injected to prevent bleeding (Fig. 583), an inverted U-shaped incision was made in the upper labial mucosa down to the alveolar bone. This extended from the right central incisor to the left first premolar. This flap was turned back, and a window was made in the alveolar bone with a gouge. This procedure exposed the large cystic cavity which was full of amber-colored fluid (Fig. 584). The fluid was aspirated, and the cavity was found to extend into the maxillary sinus. A supernumerary tooth pointing toward the nose could be seen in the central region. This tooth was removed and found to be of rudimentary shape. The cystic membrane was enucleated in one piece (Fig. 585). A window was made

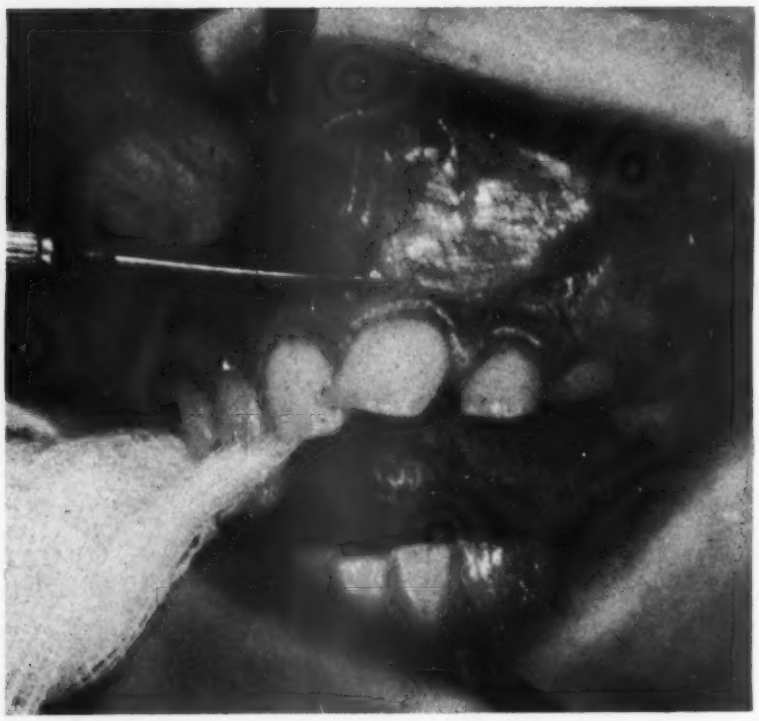


Fig. 583.—Case 128. Submucous injection of monocaine epinephrine.



Fig. 584.—Case 128. Incised cyst contained viscous fluid.

beneath the inferior turbinate bone. A Hibbs sponge was drawn through this opening from the nose several times, after which a nasal pack saturated with petrolatum was placed into the maxillary sinus and allowed to project through the nostril. The flap was sutured with dermalon sutures (Fig. 586), and the postnasal pack removed.

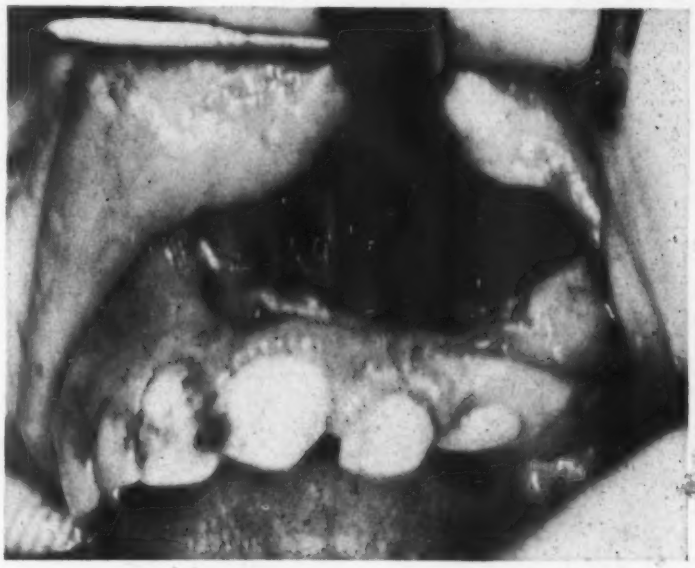


Fig. 585.—Case 128. Cystic cavity after removal of the cyst sac.

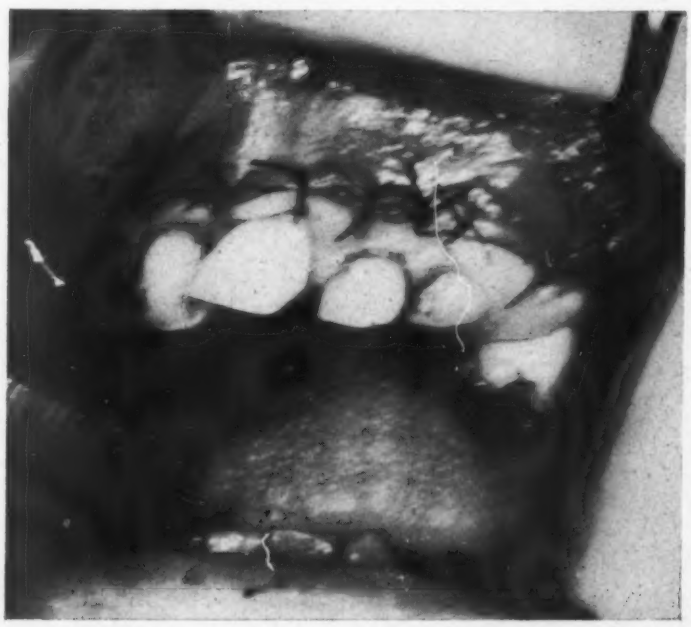


Fig. 586.—Case 128. Mucosa closed by suture.

The patient was given an intravenous infusion of 500 c.c. of 5 per cent dextrose in water after the operation. He also received penicillin, 24,000 units intramuscularly on the day of operation, and 96,000 units on each of the next four days. The white blood count was 8,400 and the hemoglobin 14.5 Gm.

On the first postoperative day there was very little swelling, and the wound was washed with warm saline. On November 9 the antral pack was removed under intravenous pentothal anesthesia. The sutures were removed on November 11, and the patient was discharged two days later. He was seen one week later in the Outpatient Department; the area was healing normally. He was last seen on December 11 when the outer wall of the maxilla was found to be still expanded, but it was felt that the deformity would correct itself in time. Nasal examination showed a patent window under the turbinate bone.

Pathologic examination : The specimen consisted of several pieces of hemorrhagic membrane, smooth and glistening, and measuring 6 by 5 by 0.5 cm. Microscopic examination revealed a cystic structure composed of a connective tissue capsule in which numerous inflammatory cells were seen. The cyst was lined by stratified squamous epithelium which in some areas penetrated deeply into the connective tissue with anastomosing cords. On the periphery of the cyst capsule there was new bone formation; the trabeculae were arranged perpendicular to the cyst wall. Diagnosis: Dentigerous cyst.



Fig. 587.—Case 129. X-ray of right side of mandible showing cyst extending into the subapical area of the first molar.

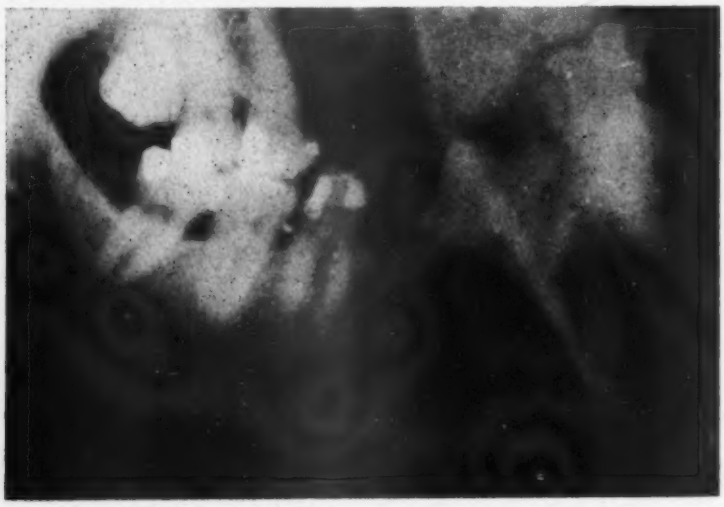


Fig. 588.—Case 129. X-ray of the cystic area extending as far as the first molar on the left.

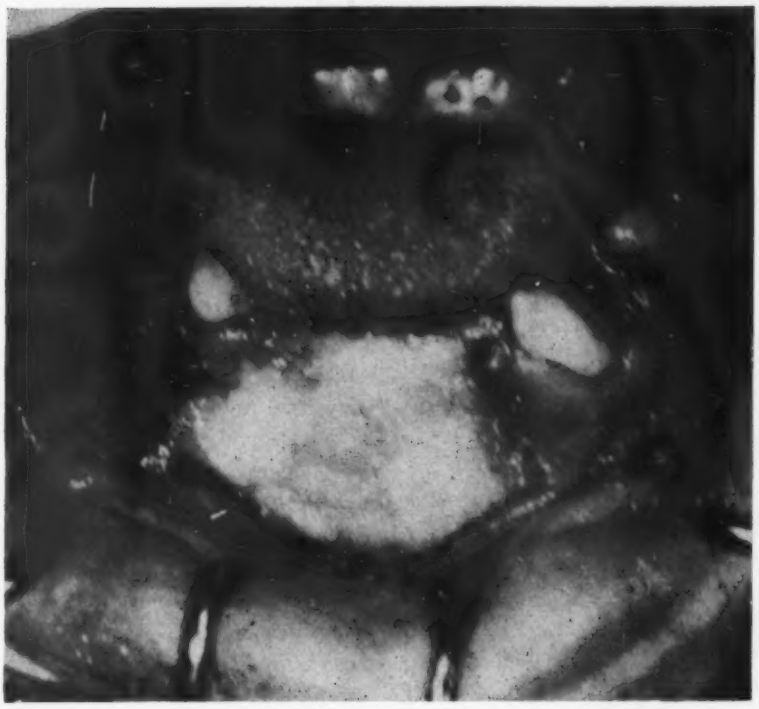


Fig. 589.—Case 129. Postoperative photograph showing dressing placed into the two pockets forming the anterior defect.

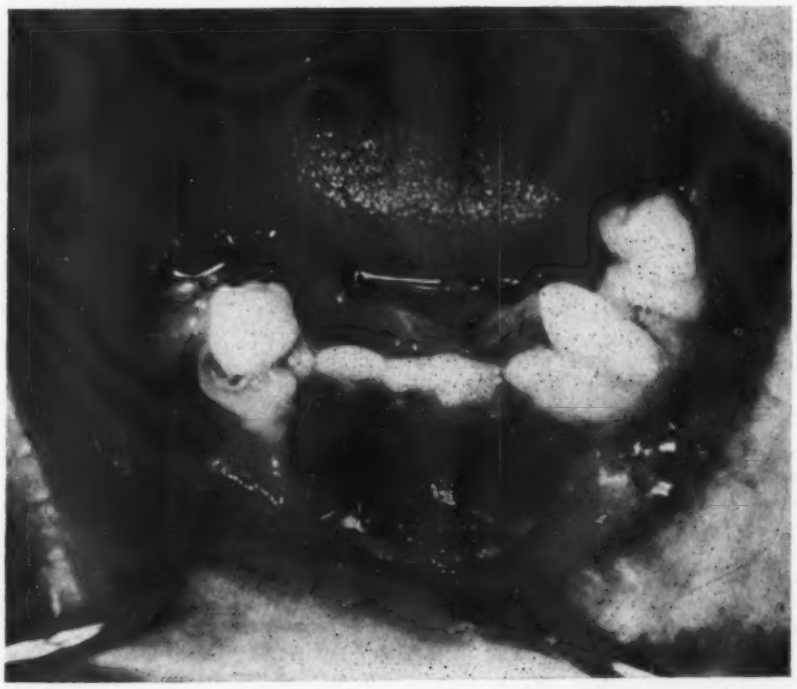


Fig. 590.—Case 129. Photograph showing condition of the wound five weeks postoperatively.

Case 129
Odontogenic Cyst of Mandible

H. W. (513312), a 42-year-old man, was admitted to the hospital on Dec. 6, 1945, with a diagnosis of adamantinoma of the jaw. The lesion was noted six weeks ago at a routine roentgen examination by the patient's dentist. There have been no symptoms.

Examination showed the lower right first premolar and canine to be tilted anteriorly. The upper right third molar and the upper left canine were impacted, and the deciduous canine was in place.

X-ray examination revealed a cystic lesion of irregular outline and a poorly defined margin. It extended approximately from the first molar on the left to the first molar on the right, causing a thinning of the cortex of the inferior border and a displacement of the canine and second premolar teeth on the left (Figs. 587 and 588).

On December 7, under intratracheal gas, oxygen, and ether anesthesia, an incision was made across the gingival margin of the lower incisor teeth, and extended vertically at the ends so as to retract a mucoperiosteal flap to expose the entire mental prominence of the jaw. The bone there was extremely thin, and at one place where a perforation was made, a creamy substance escaped. The thin bone wall was removed, and a cyst sac exposed which was filled with creamy cholesterol. The sac was detached from the bone and was found to be connected with the four incisor teeth. These teeth were extracted. The dissection was carried along on each side into the area extending underneath the molar and premolar teeth. The sac was removed in one piece, and the cavity inspected. It was found that the root tips of the right canine and left premolar extended into the cavity. These two teeth were extracted. The bone edges were smoothed with files and rongeurs, and sulfanilamide powder, 4 Gm., was inserted into the wound. The mucoperiosteal flap was placed into the bottom of the bone cavity, and the wound was packed with boric strips (Fig. 589).

Two days after the operation the dressing was removed and the cavity irrigated with saline solution. There was some capillary bleeding after the dressing was taken out. The next day the sutures were removed. The patient was discharged on December 13, to be followed at the office. He made an uneventful recovery, and after five weeks he was given a syringe to irrigate the wound until it was completely healed (Fig. 590).

Pathologic report: A spider-shaped piece of tissue measuring 6 by 3 by 0.4 cm. was received. It had a smooth white surface on one side and a hemorrhagic uneven surface on the other. Microscopic examination revealed a cyst composed of a rather dense collagenous connective tissue capsule in which there were many inflammatory cells, chiefly plasma cells, lymphocytes, and histiocytes. Many Russell fuchsin bodies were noted. Hemorrhage was present in several areas of the capsule. The cyst was lined almost completely with stratified squamous epithelium which in some areas was markedly thickened. Diagnosis: Odontogenic cyst.

Case 130
Giant-Cell Tumor of the Mandible

L. B. (507367), a 7-year-old girl, came to the dental clinic on Oct. 11, 1945, with a swelling on the gingiva in the lower incisor region.

This swelling was first noticed about two and one-half months before when the lower deciduous incisors were shed, and when the permanent teeth erupted. She was recently referred to the hospital by the school dentist.

Examination showed a firm mass, 2 by 3 cm., extending over the occlusal surface of the teeth in the region of the lower left lateral incisor. It extended to the mucobuccal fold labially, and to the floor of the mouth lingually. It was firmly attached with a wide base and appeared to involve the bone. The mass was rather cyanotic in appearance and not ulcerated (Fig. 591). It did not bleed on palpation, but bled when the patient ate.

X-ray examination showed a large area of bone resorption around the unerupted lower left second incisor extending to the region of the central incisor. The deciduous lateral incisor had been lost (Fig. 592). Numerous carious teeth were present.

Diagnosis: Giant-cell tumor.

The patient was admitted to the House on October 24. The white cell count was 7,500 and the red cell count 4,200,000. Two days later excision of the tumor was performed under endotracheal, gas, oxygen, and ether anesthesia. The anesthesia apparatus was removed while the electrocoagulation was being used, and the posterior part of the mouth and throat was packed with wet gauze. After the usual preparation of the mouth, the incision was



Fig. 591.—Case 130. Photograph showing a purplish tumor mass attached to the anterior part of the alveolar process.

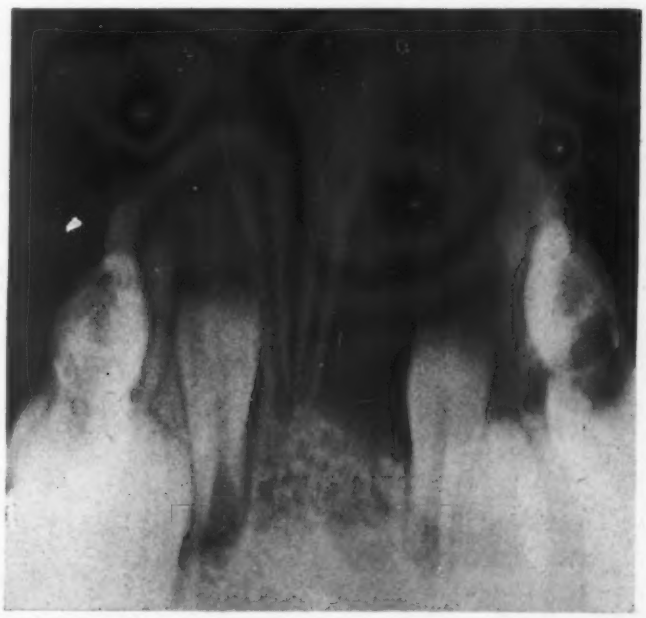


Fig. 592.—Case 130. X-ray showing an area of resorption around the unerupted second incisor.

made with the endothermy knife (Fig. 593). The tumor was excised along with the lower right central incisor and unerupted lateral incisor. It had a brownish appearance. The tumor bed was swabbed with phenol and alcohol. A boric strip was inserted into the cavity and two sutures were used to hold it in place.

On the first two postoperative days the patient had an increased temperature, but the oral wound appeared normal and was painted with gentian violet. She was given 96,000 units penicillin intramuscularly on the third postoperative day, and on each of the next four days. Recovery was uneventful and the patient was discharged on November 2. She was seen several times in the Outpatient Department, where the mouth was sprayed out, and the wound was found to be healing normally. She was discharged on Jan. 30, 1946, completely recovered, with no evidence of recurrence (Fig. 594).



Fig. 593.—Case 130. Excision of tumor by means of electrocautery.



Fig. 594.—Case 130. Result three months postoperatively.

Pathologic report: The specimen consisted of a nodule of soft, dark red-gray tissue measuring 2 by 1 by 1 cm. It was covered on two sides by pearl gray epithelium. Microscopic examination showed a section covered by stratified squamous epithelium with marked

parakeratosis. The main portion of the tumor consisted of connective tissue composed of round to spindle-shaped fibroblasts with a moderate amount of collagen. Throughout the section there were numerous multinucleated giant cells which varied in size and shape. Some of these cells contained as many as 35 nuclei (Fig. 595). Hemorrhage was evident in many areas. On one edge there was a spicule of bone which seemed to be undergoing resorption. Diagnosis: giant-cell tumor.

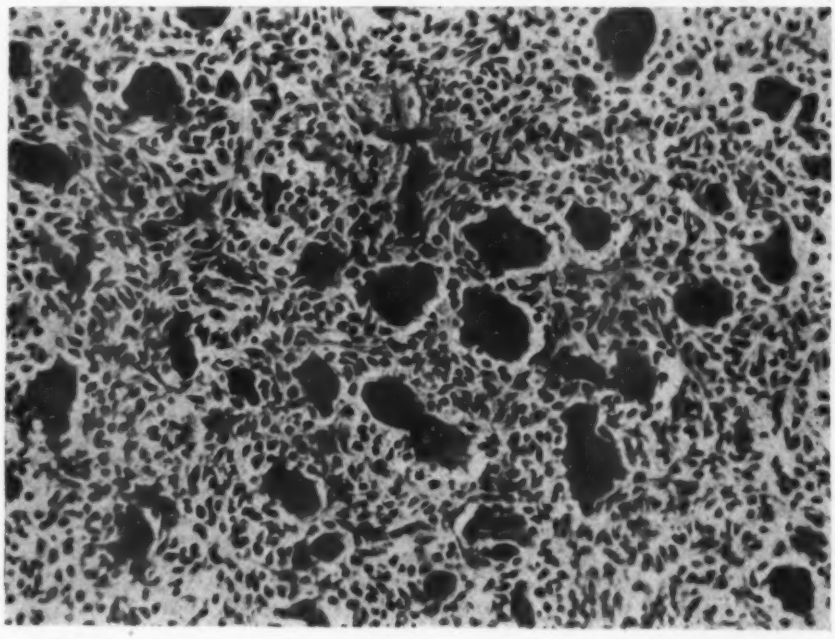


Fig. 595.—Case 130. Photomicrograph of section which was diagnosed as giant-cell tumor.

Case 131

Myxoma of Mandible

R. G. (517071), a 24-year-old man, entered the hospital on Jan. 15, 1946, with a tumor of the mandible.

Three years before, the patient noticed a swelling on the jaw in the region of the lower left premolars. It gradually increased in size but was not painful. One and one-half years before, a cyst had been excised, the membrane removed, and the area sutured. The swelling did not decrease in size, and has gradually increased since then.

Examination showed an egg-sized, hard, smooth tumor on the mandible, extending from the left second premolar to the midline. The jaw bulged both labially and lingually about ¼ inch. The lower left canine, lateral and central incisors were loose. The lower left first premolar was missing. A scar of a previous excision was present.

X-ray examination showed a multicystic tumor expanding the mandible (Fig. 596), and extending from the right first premolar to the subapical area of the left first molar (Fig. 597). Impression: (1) adamantoblastoma; (2) odontogenic cyst.

On January 16, under endotracheal gas, oxygen, and ether anesthesia, a partial resection of the mandible was performed. An incision was made on the outer surface of the alveolar process close to the gingival margin extending from the left second molar to the right first molar. Vertical incisors were added on each side and in the center. The mucosa was carefully dissected away from the bone as far as the inferior border of the mandible. At the left side the mental foramen was exposed and the nerve preserved. The same was done on the right side. The dissection was then carried along underneath the chin. The muscles attached to the mental prominence were cut, and the bulging bone containing the

entire tumor mass became visible. Healthy bone was seen at the distal ends on each side. By means of a drill the bone was incised in the healthy area on each side. This vertical incision was carried along horizontally very close to the inferior border of the mandible. The thin bony wall was removed on the outer surface so as to expose the tumor, which had a white, glistening color, and was of a solid, jellylike consistency with no cystic fluid (Fig. 598).

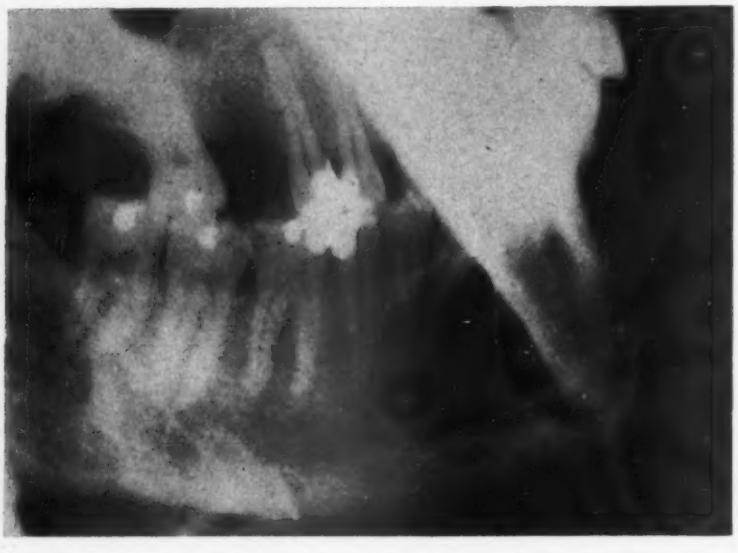


Fig. 596.—Case 131. X-ray of the right side of the mandible showing a polycystic lesion with marked expansion of the bone.

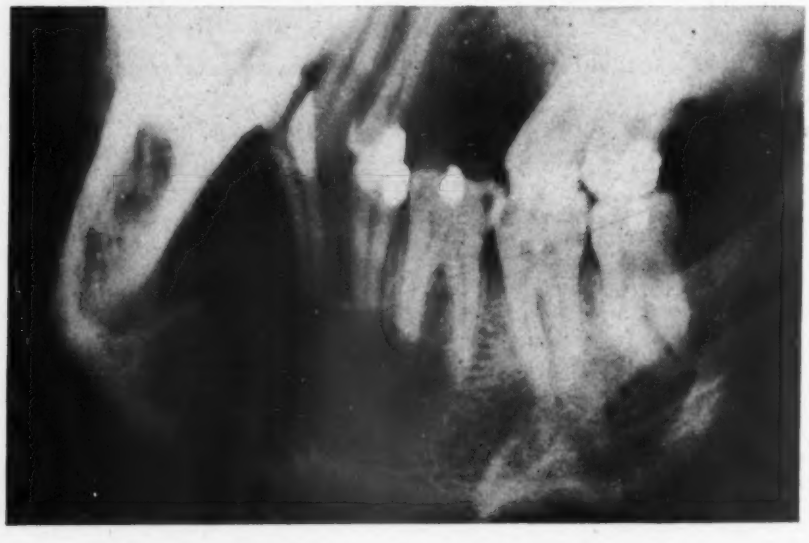


Fig. 597.—Case 131. X-ray of left side of the mandible showing the lesion extending below the first molar tooth.

An incision was then made on the lingual surface of the elveolar process, and all the teeth from the left lower second molar to the right lower second premolar were loosened from their posterior attachment, and the tumor removed in one piece with the teeth in situ, except for the two last teeth which tore away when the tumor was removed (Fig. 599). Examination of the resulting bone cavity showed the inside of the lingual surface of the mandible. The mandibular nerve was exposed on the left, and its branch formerly trans-



Fig. 598.—Case 131. Photograph showing the exposure of the tumor mass after the muco-periosteal flap was retracted and the bone removed.



Fig. 599.—Case 131. Photograph showing the tumor being removed with the teeth in situ.



Fig. 600.—Case 131. Photograph showing the lingual bone with fracture wired by the transosseous method.

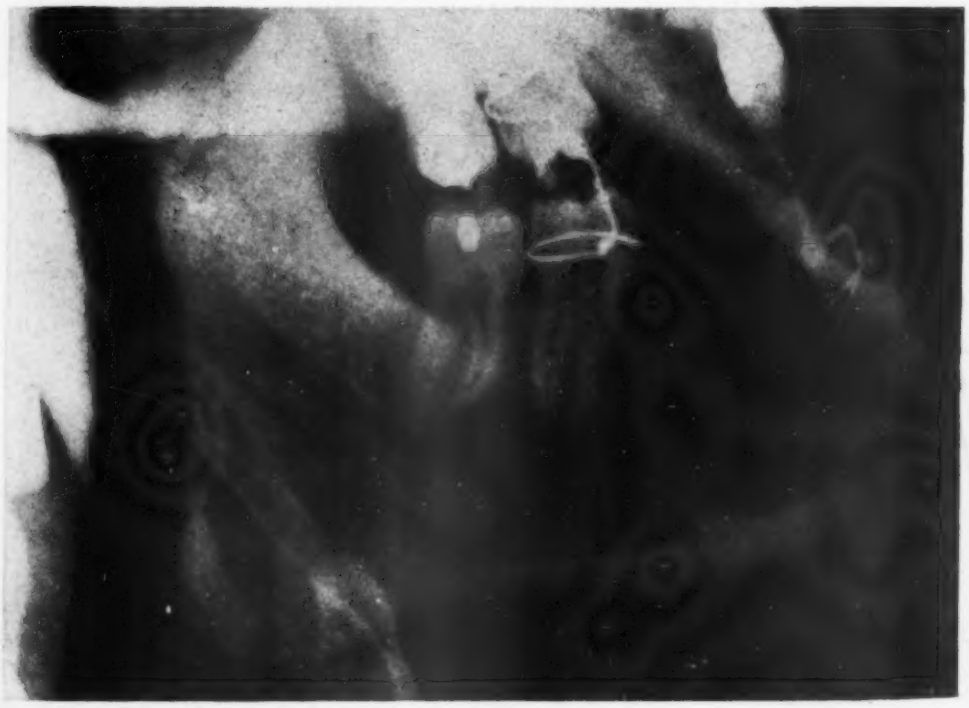


Fig. 601.—Case 131. X-ray showing the postoperative result and wire suture in place.

fixed by the mental foramen was laid free so that there would be no numbness of the lip. The bone was very carefully inspected for evidence of any remaining tumor or any extension into the accessory cavity. On the left side of the symphysis, where the x-ray had shown invasion of the bone, the entire part was removed. On the right, the tumor cavity extended

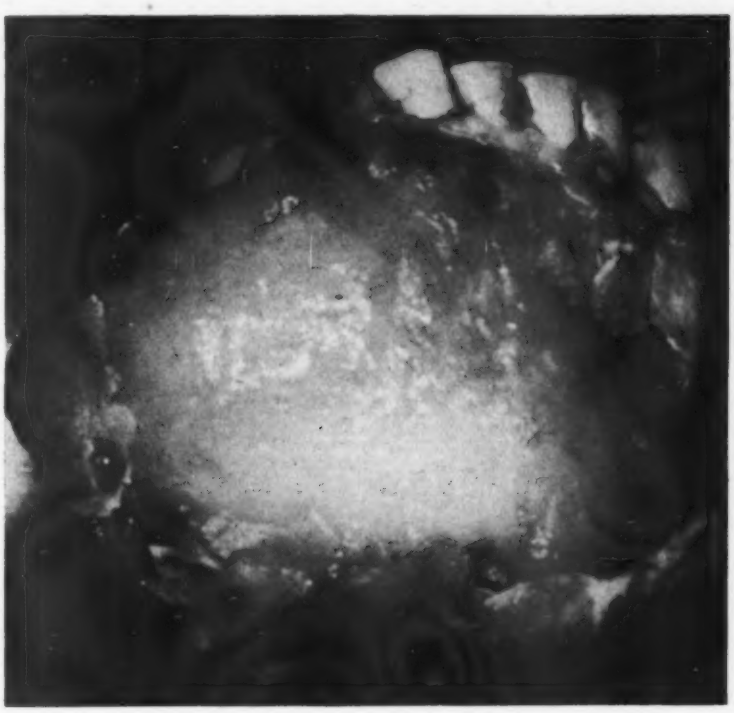


Fig. 602.—Case 131. Labial aspect of the excised tumor.



Fig. 603.—Case 131. Lingual aspect of excised tumor.

under the second premolar. It was necessary therefore, to extract this tooth. It was firmly embedded, and in spite of chiseling the bone away on the outer side, considerable force was needed to extract it. This fractured the jaw at the symphysis where the bone was very thin. A hole was drilled in the remaining part of the lower border at this place in each fragment,

and a stainless steel wire was passed through and twisted on the inner surface of the bone for transosseous fixation. The bone was well impacted and held firmly by wire sutures (Fig. 600). The bleeding from the mandibular canal continued on both sides, and fibrin foam and thrombin were applied to the bleeding areas. The mucosa, which had contracted on the lip, was then stretched out and sutured to the lingual mucosa, and a drain was inserted into the bone cavity to take care of any bleeding that might occur. Because of the fracture, it was thought best to immobilize the mandible. This was done by intermaxillary ligation on the remaining molar teeth. A good occlusal result was obtained. The patient left the operating room in good condition, after an elastic bandage had been applied.

During the operation the patient received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water; this was followed later with 1,500 c.c. of 5 per cent dextrose in saline. He also received 64,000 units of penicillin intramuscularly on the day of operation, 96,000 units on the first postoperative day, and 60,000 units on the second postoperative day plus penicillin lozenges every hour. There was no postoperative pain or bleeding. The drain was removed on the second postoperative day.

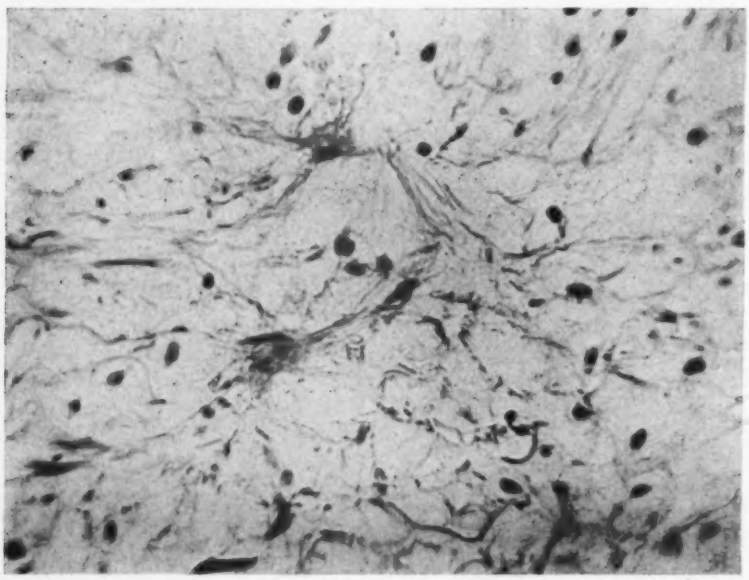


Fig. 604.—Case 131. Photomicrograph of section showing tumor. Diagnosis: myxoma.

X-ray examination on January 19 revealed a large defect involving the symphysis of the jaw at the site of the removal of the tumor. The defect had a sharply defined margin, and there was no evidence of infiltration by the tumor. A wire suture was seen holding the fracture at the symphysis (Fig 601).

The patient was discharged on January 21, to be followed at the office.

Pathologic examination: The specimen consisted of a portion of the lower jaw, measuring 4 by 4 by 2 cm. with seven teeth contained in it. There was complete destruction of the bone and replacement by a homogenous soft tissue extending to both cut edges of the specimen, and it did not appear to be encapsulated (Figs. 602 and 603). Microscopic examination revealed a polypoid mass composed of myxomatous tissue. In some areas the cells appeared to have long spindly processes. Sprinkled throughout the tissues were lymphocytes and a few plasma cells and large mononuclear cells. Diagnosis: fibromyxoma (Fig. 604).



Fig. 605.—Case 132. Photograph showing patient with tumor mass at the angle of the jaw.



Fig. 606.—Case 132. Intraoral view showing a polypous mass attached to the lingual side of the alveolar process.

Case 132

Adamantoblastoma of the Mandible

T. C. (509461), a 46-year-old woman, was admitted to the hospital on Nov. 2, 1945, with a tumor of the left mandible.

Four years before, the patient had had a tumor removed which was diagnosed as adamantoblastoma. This had recurred and the patient was referred here by her local dentist. A biopsy made a few days before entry was also diagnosed as adamantoblastoma.

Examination by palpation showed a swelling of the left mandible about 10 by 10 cm. in size, a firm, smooth mass firmly attached (Fig. 605). It was nontender, and there were no palpable nodes. Intraorally there was a redundant piece of tissue seen extending from the posterior part of the gingiva of the lower left mandible (Fig. 606).

X-ray examination revealed an osteolytic central defect at the angle of the jaw (Fig. 607). There was also evidence of resorption at the posterior part of the inferior and superior borders of the jaw, extending well into the ascending ramus.

Diagnosis: Adamantoblastoma of the mandible.

On November 5 a resection of the left mandible was performed under intratracheal gas, oxygen, and ether anesthesia. The patient's blood was typed so that a transfusion could be given during the operation if necessary.

First, a vertical, angulated incision was made in the preauricular area. Several vessels were tied, the zygoma was exposed, and the mandibular joint located. The condyle was dissected down upon. The periosteum was incised, and two condylar retractors inserted beneath it. With an electric drill, an osteotomy was performed in the subcondylar region. The subcutaneous tissue was then closed with catgut, and the skin with interrupted dermalon sutures. The condyle so detached was to serve later as an attachment for a bone graft.

A curved incision was then made below the inferior border of the mandible, starting at the anterior border of the sternomastoid muscle and extending forward to the mental region (Fig. 608). The platysma was divided. The external maxillary artery and vein were ligated, and the angle of the jaw exposed. At this point a large tumor mass was visible extending from the angle of the jaw down to the inferior border. This mass was dissected away from the skin flap and the outer surface of the ramus exposed. The dissection was then continued forward until normal bone was found in the horizontal part of the mandible. Here the mandible was divided by means of a Gigli saw (Fig. 609). By blunt dissection, the posterior part of the mandible was detached from the underlying structure, the internal pterygoid muscle and the stylomandibular ligament. The inner surface of the ramus could now be inspected, because tumor masses were found to extend from here also. The mucosa of the mouth was then incised, leaving the small tumor masses extending into the oral cavity attached to the jaw. The dissection was carried on until the coronoid process was finally felt, and its tip divided by means of bonecutting forceps. The mandible was now attached only to the sphenomandibular ligament and the mandibular artery and nerve, which could not be seen because of the tumor mass. Both the nerve and artery were torn when removing the specimen, and a considerable amount of hemorrhage immediately ensued. This was first arrested by pressure, until finally the bleeding vessel could be found; it was the internal maxillary artery. It was tied on both ends. The wound was then inspected. There was no tumor visible (Fig 610).

The mucosa was sutured to close the opening into the mouth (Fig. 611). A clear acrylic temporary prosthesis, which had been previously prepared to fit between the condyle and the osteotomized mandible, was inserted to prevent scar contraction and displacement of the jaw. It was found to fit accurately. Two holes were drilled in both the prosthesis and the mandible, through which 24 gauge stainless steel wire was passed, attaching the prosthesis to the bone in the anterior part of the mandible (Fig. 612). In the condylar region it was left unattached. The wires were bent so they would not irritate the soft tissues, the subcutaneous tissue was closed with catgut and the skin with interrupted dermalon sutures (Figs. 613 and 614).

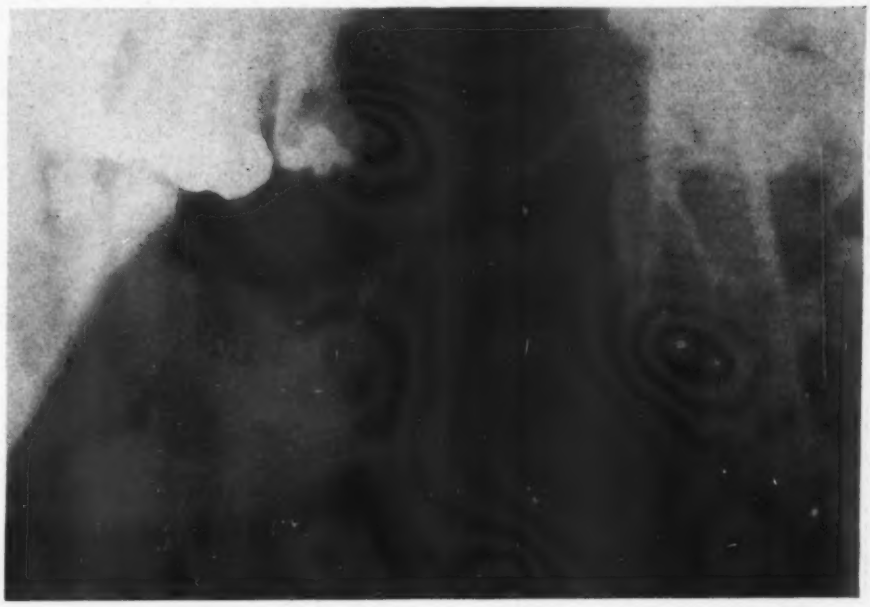


Fig. 607.—Case 132. X-ray showing a cystic cavity; with bone destruction at the anterior border of the ascending ramus and the angle of the jaw.



Fig. 608.—Case 132. Photograph taken after the osteotomy at the base of the condyle had been completed and the incision sutured. An incision was then made from the posterior surface of the angle of the jaw, extending 1 cm. below the inferior border as far as the chin.

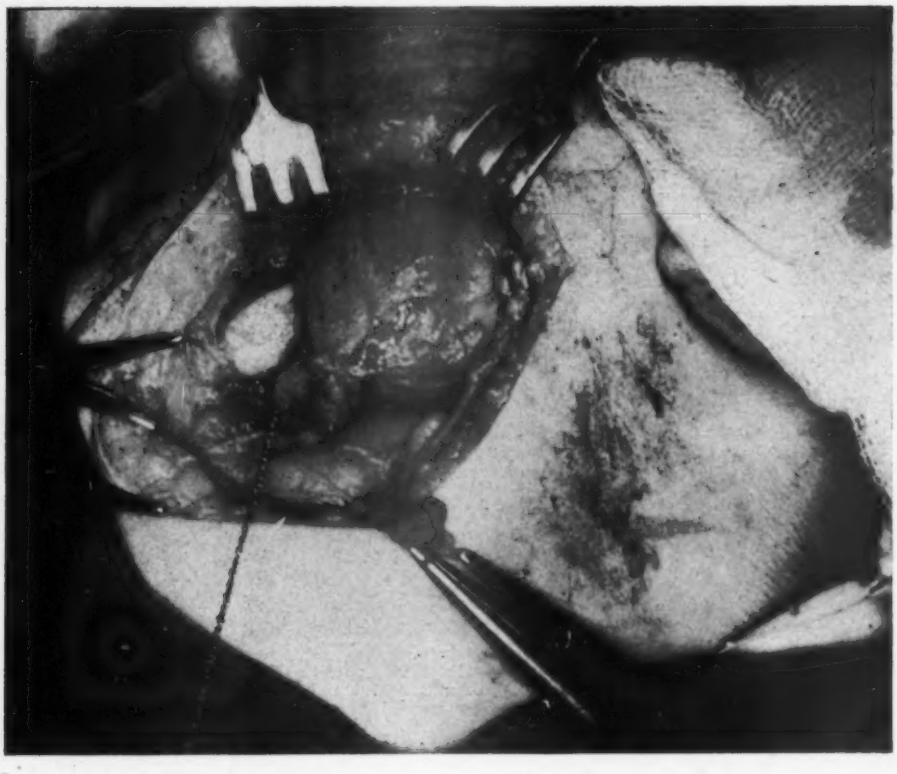


Fig. 609.—Case 132. Photograph showing the tumor exposed and an osteotomy being performed with a Gigli saw behind the mental foramen.



Fig. 610.—Case 132. Tumor in mandible excised with part of the buccal mucosa, and perforating the oral cavity, so that the tongue (*T*) is seen. *B*, The anterior part of the mandible.



Fig. 611.—Case 132. Suturing of buccal mucosa.

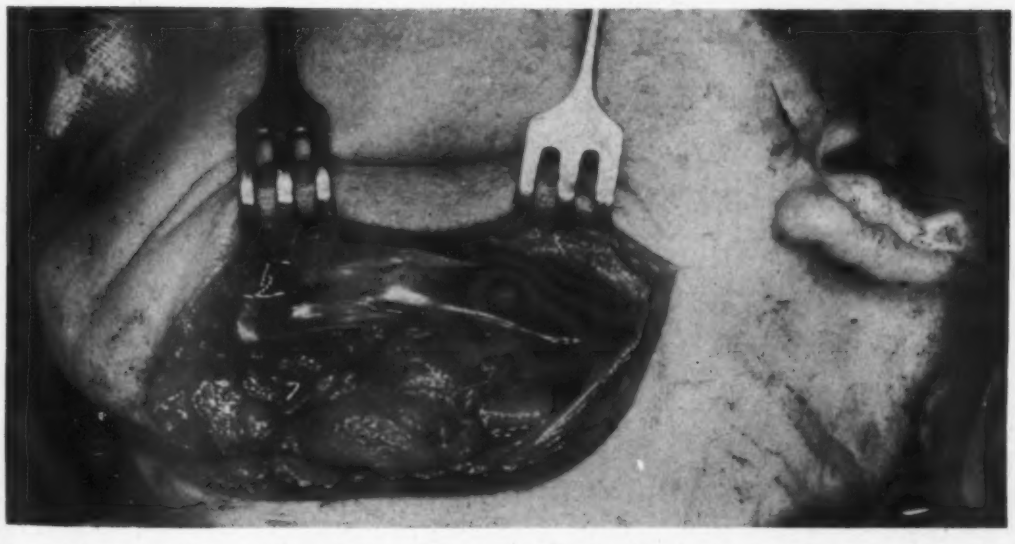


Fig. 612.—Case 132. Acrylic splint was inserted and attached by two stainless steel wires to the anterior part of the mandible.

Preoperatively the patient received 36,000 units of penicillin intramuscularly as an initial injection, and 12,000 units every three hours thereafter. On each of the next three postoperative days she received 96,000 units, and 120,000 units on the following three days. She was given a transfusion of 500 c.c. of whole blood during the operation, and later, an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water.



Fig. 613.—Case 132. Closing the subcutaneous tissue over the splint with interrupted catgut sutures.



Fig. 614.—Case 132. Skin sutures completed, closing the incision.

One the first postoperative day there was some swelling of the face and ecchymosis of the left eye. The intraoral wound was painted with gentian violet. On the sixth postoperative day the sutures were removed.

On November 13 the x-ray examination showed a large portion of the left mandible removed. There was no evidence of osteomyelitis in this region. The condyle was all that remained of the distal portion of the mandible.

On November 14 there was some sloughing intraorally in the area of the wound. The oral incision had opened and the slough increased considerably, so that the prosthesis could be seen. It was felt that this was due to too great a strain on the sutured mucosa during mastication, and also perhaps because catgut was used; a dermalon suture might have been better. On November 17 the anterior end of the skin incision was opened up and a large

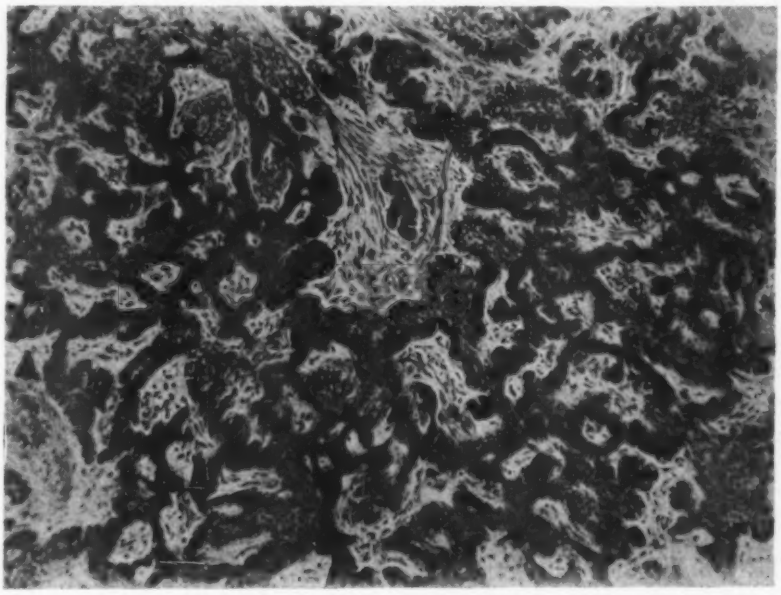


Fig. 615.—Case 132. Photomicrograph showing epithelial arrangement of adamantoblastoma.

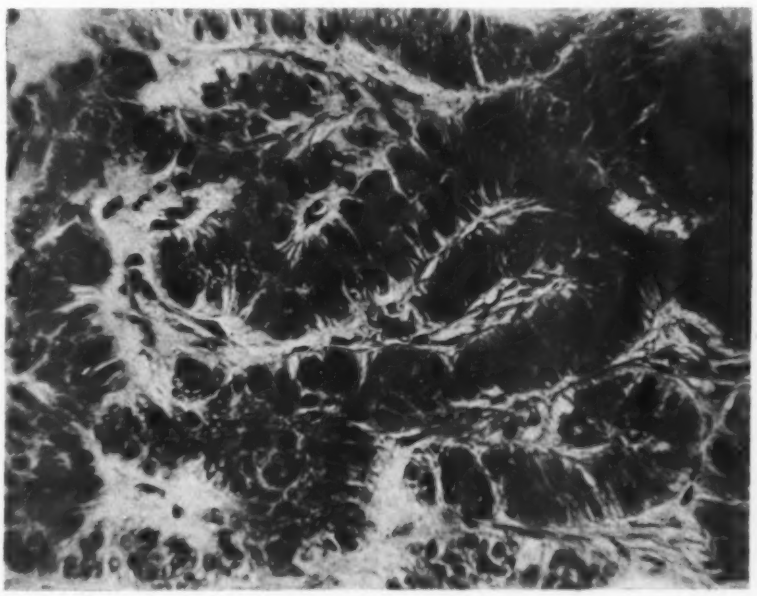


Fig. 616.—Case 132. Photomicrograph showing polyhedral cells making up the tumor.

amount of fluidlike substance resembling necrotic tissue was removed, after which a dry sterile dressing was applied. The mouth was painted with zephiran. Penicillin therapy was again started on November 14, and 96,000 units were given intramuscularly each day.

The drainage of fluid from the wound persisted, and it was irrigated daily. It was

felt that because the wound opened, the prosthesis was acting as a foreign body. On November 24 the prosthesis was therefore removed under intravenous pentothal sodium anesthesia. This was accomplished by enlarging the intraoral incision at the anterior end, after which the two wires holding the prosthesis could be cut and removed. The prosthesis was then easily withdrawn. The edges of the entire buccal perforation were freshened and the wound sutured with dermalon sutures from the oral side. A Dakin tube was inserted through the fistula which had formed in the skin incision, and was attached to the skin edges with dermalon sutures. The patient was given an intravenous infusion of 1,500 c.c. of 10 per cent dextrose in saline.

Three cubic centimeters of penicillin, 500 units per cubic centimeter, were run into the Dakin tube four times a day. Two days later the tube was removed. Intramuscular injections of penicillin were discontinued on December 2. Two days later the intraoral sutures were removed, and the patient was discharged on December 5 to be followed at the office. She was referred to a specialist on surgical prothesis for the construction of an appliance to control the remaining part of the jaw.

Pathologic report: A segment of mandible 7 cm. long was submitted. The angle and adjacent ramus and body were surrounded by a firm, irregular, pinkish-gray mass, 5.5 cm. long and 3 to 4 cm. in diameter on cross section. The tumor had eroded almost completely through the body just proximal to the angle. It approached to within 2 mm. of the proximal resection margin and to within 1 cm. of the distal end of the ramus along its outer surface. When the tumor was divided, the cut surfaces were glistening gray and divided into irregular nodes of thin fibrous bands.

Microscopic examination showed large lobules of interlacing cords (Fig. 615), and various-sized aggregates of round to polyhedral epithelial cells (Fig. 616). In some areas peripheral cells of the lobules were columnar in shape, with a centrally placed nuclei. The central cells appeared rather stellate in some instances, and square in others. These columnar cells and the type of nuclei identify the tissue as adamantine epithelium. The cords and lobules resembled the tooth bud stage. In two areas the adamantine tissue was proliferating against muscle tissue, and in another, bone was seen approximating the tumor. The matrix consisted of fibrous connective tissue, which was somewhat stellate in appearance. Many blood vessels were noted. Diagnosis: Solid adamantoblastoma.

The patient went home soon after the completion of the appliance to control the jaw. She reported on March 10 that she was well and working, and could eat well. She will return for a bone graft later.

Case 133

Carcinoma of the Maxilla

L. G. (509574), a 40-year-old woman, was admitted to the hospital on Nov. 19, 1945, with a diagnosis of carcinoma of the left maxilla.

About one and one-half months ago the patient noticed a small lesion on the left side of the hard palate which she believed to be a canker sore. It did not improve with time, however, and she sought treatment.

Examination showed an ulcerated lesion which extended from the third molar region to the second premolar in the left maxilla, and to the midline of the hard palate (Fig. 617). The ulceration could be seen in the interdental papilla of the molar from the buccal aspect. There was leucoplakia of the cheeks and lower alveolar ridges, and papillary atrophy on one side of the tongue. The four upper anterior teeth had been lost and replaced with a fixed bridge. A biopsy showed carcinoma.

X-ray examination revealed destruction of the maxilla in the region of the extracted first molar. The x-ray of the sinuses showed a rounded cystlike mass in the floor of the right antrum. There was no significant thickened membrane in any of the sinuses. Lateral views showed a rounded shadow of soft tissue density overlying the anterior portion of the maxilla. The anterior wall of the antrum was poorly defined in this projection, and suggested bone destruction.



Fig. 617.—Case 133. Photograph showing ulcerative lesion on the hard palate, which was found to be carcinoma.

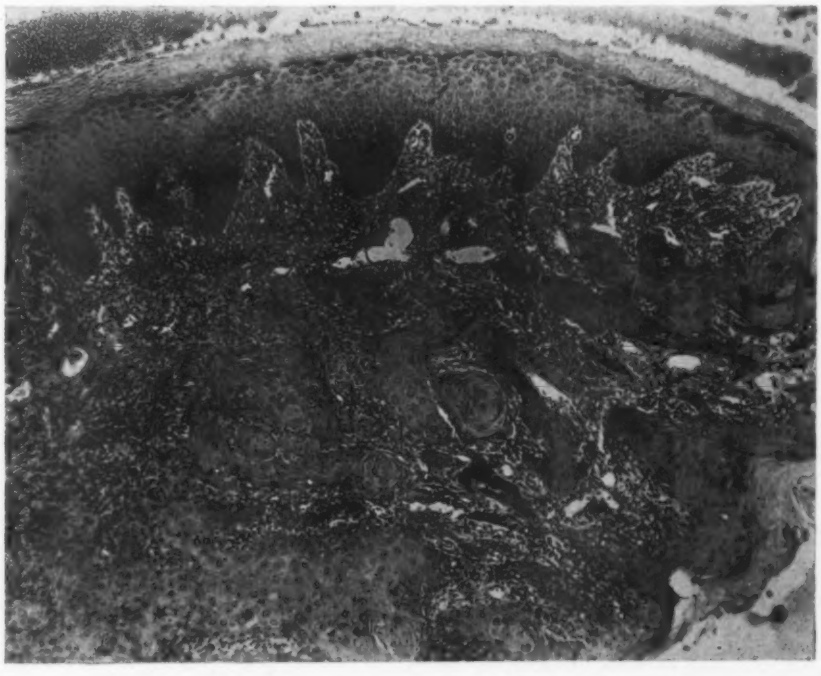


Fig. 618.—Case 133. Photomicrograph showing epidermoid carcinoma, grade II.

On November 21 the carcinoma was destroyed by fulguration. Previous to operation the patient's blood was typed, and one blood setup was on call at the operation. Under pentothal sodium intravenous anesthesia with an intratracheal tube inserted through the nose and a postnasal plug to close the passage, the teeth on the left side of the maxilla were extracted. An incision with the endothermy knife was made in the mucosa, leaving a 0.5 cm. margin of healthy tissue. The palate was fulgurated and the carcinoma removed with a loop. The bone was then coagulated and removed with rongeur forceps until there was a wide perforation of the maxillary sinus. The margin of the wound was coagulated for 1 cm. all around. Bone bleeding was stopped by coagulation, and the bony walls smoothed. A piece of boric strip was placed into the maxillary sinus.

During the operation the patient received an intravenous infusion of 1,500 c.c. of saline with one ampule of metrazol. She was given 96,000 units of penicillin intramuscularly on each of the next five postoperative days, and a high-vitamin, high-calorie liquid diet.

On the second postoperative day the dressing was removed, and petrolatum, iodoform gauze inserted. The wound was painted with 1 per cent aqueous gentian violet. The dressing was changed daily, and on November 27 it was removed. The patient was discharged on November 28 to be followed at the office. She was given penicillin troches* to control the odor from the slough. After ten days the slough separated. The patient was comfortable. Pieces of bone were removed from time to time, and finally the wound showed a healthy appearance.

Pathologic examination: Microscopic examination showed a polypoid lesion covered on one side by stratified squamous epithelium showing marked parakeratosis. In the center the epithelium was greatly thickened and infiltrated deeply into the submucosa. Islands of epithelium showing marked anaplasia were present in the connective tissue. The cells showed bizarre forms and many mitotic figures. Keratinization, whorls, and pearl formations were present. There was marked inflammatory infiltration consisting of lymphocytes, plasma cells, and large mononuclear cells. Mucous glands were seen at the base of the lesion. Diagnosis: Epidermoid or squamous-cell carcinoma, Grade II (Fig. 618).

*Supplied for experimental purpose by the Schenley Company.

GINGIVECTOMY: INDICATIONS, CONTRAINDICATIONS, AND METHOD

HENRY M. GOLDMAN, D.M.D.

THE treatment of periodontal disease without regard to diagnosis, etiology, and pathology is apt to result in failure. Therefore, if we are to treat our cases successfully, we must have a thorough knowledge not only of the physiology of periodontal structures, but also of the changes seen in the disease of these tissues. Combined with an understanding of the etiological factors, treatment is successful.

OBJECTIVES

One of the ultimate aims in periodontal treatment is the elimination of the pathologically deepened gingival crevice. This may be accomplished by various methods, one of which is gingivectomy. This may be defined as the surgical removal of the gingiva to the bottom of the pocket. Ascertainment of the depth and shape of the pocket and its relationship in position to the alveolar crest is of primary importance. Some operators believe that in periodontal disease the bone crest is infected or necrotic and, therefore, should be removed when a gingivectomy is performed. This theory is not substantiated by histologic evidence, since the bone is never necrotic as it is in osteomyelitis. If the gingivectomy is performed correctly, no bone can be exposed since only the free margin of the gingiva is removed. Fig. 619 shows the interdental tissue between the first and second mandibular premolars. Calculus deposits are seen at *C*, alveolar bone at *B*, and the bottom of the pockets at *E*. The dotted line demonstrates the extent to which the gingiva should be removed in gingivectomy.

INDICATIONS

If a pocket is so shaped or situated that it will not disappear when the gingival tissues shrink after subgingival curettage, gingivectomy may be employed. It should also be used in cases where clinical reattachment cannot be obtained, or in cases of gingival hyperplasia where the tissue is not reduced by curettage and stimulation. Here the operation may be termed gingivoplasty rather than gingivectomy. Gingivectomy may also be used in cases where the interdental gingiva is destroyed and the buccal and lingual gingiva remain intact.

CONTRAINDICATIONS

Gingivectomy should not be employed in those cases where the crevices are shallow and the gingiva is swollen due to inflammation. Here it is usually not



Fig. 619.—Interdental tissue between the first and second mandibular premolars. *C*, Calculus; *B*, alveolar bone; *E*, bottom of pockets. (U. S. Army Medical Museum Neg. No. 78386.)

necessary to remove tissue since the gingival margins will regress when the inflammation subsides and resolves after subgingival curettage, gingival stimulation, and mouth rehabilitation. Gingivectomy is also contraindicated in those instances in which the base of the deepened gingival crevice is placed further apically than the alveolar crest. In such intrabony pockets, an attempt to produce reattachment can be made. It is necessary that all the epithelium in the pocket be removed, and the tissue readapted. The blood between the tooth and bone is allowed to clot, and should be protected by suturing the tissue or covering with a pack. If the operation is successful, the blood clot organizes into connective tissue, and a layer of cementum is formed on the root of the tooth in which periodontal fibers are incorporated. This is also true of the bone. The depth of the crevice will then end at the point of formation of new tissue (connective tissue, cementum, and bone).

METHOD

Before gingivectomy, the teeth should be thoroughly scaled and all the calculus and local irritations removed. The operation should be performed under local anesthesia. The field should be prepared aseptically with a solution of castile soap and sponged with an antiseptic solution. The depth of the pockets, previously investigated, should be marked with either a sterile indelible pencil or by drawing a scalpel lightly over the gingivae, producing a slight incision. The incision is then made in a direction that simulates a normal gingival attachment, that is, apically away from the tooth, as otherwise a shelf will be created where food can lodge. The cut should be made at the base of the pocket, and the gingiva from around the tooth removed. This is best done by means of a small knife, or in some cases, with sharp-pointed scissors. The incisions should be smooth and clean and the tissue should not be traumatized.

If gingivectomy is to be employed throughout the mouth, the buccal incision is made first and the tissue lifted off. The lingual cut is then made, using the knife at an angle that results in the correct bevel. The lingual gingivae are then raised. With a small knife that can pass through the interproximal spaces, the interproximal tissues are severed buccopalatally or buccolingually. The entire tissue is removed. The interproximal tissue then may be curetted, and all inflammatory granulation tissue removed. No ragged tissue or tabs should remain. Some operators used the electrocautery to remove these tabs of tissue, and it may also be employed to festoon the gingivae. In many instances it is helpful in trimming the interproximal tissues.

After the operation, bleeding should be stopped; this is best done by packing the interproximal spaces with small pieces of cotton saturated with adrenalin or any hemostatic preparation. After the bleeding has stopped, a pack should be placed on the wound surface. This pack should be mixed very stiff so that it is the consistency of putty; it is placed between the teeth and packed over the gingivae. It should extend from the buccal to the lingual surface, and interproximally below the contact points. It will be sufficiently firm to stay in place a few days. Upon removal, the mouth is irrigated with an astringent

solution and washed with an antiseptic. In some instances, a few areas should again be covered, and a periodontal varnish or paraffin wax may be used. The patient is instructed to keep his mouth clean.

A few days later, the mouth should again be cleaned of soft deposits by spraying. If any calculus is seen, it should be removed. When the gingivae appear fairly well healed, the teeth should be cleaned and well polished. The final result should produce healthy, pink, firm gingivae with complete elimination of the pockets.

As stated previously, the alveolar bone should not be disturbed. Curettage of the alveolar crest is not only unnecessary but erroneous in most cases. There is only one type of case where there is sufficient reason for removing alveolar bone, and this is the case where an interproximal crater is formed due to loss of bone. The buccal and lingual crests form ridges which tend to hold the food in the crater and cause further inflammation of the tissue. It may be necessary to remove this bone to obliterate this hollow space. In most instances, however, the removal of the buccal and lingual gingivae alone is sufficient to eliminate crater formation.

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DEVELOPMENT OF THE HEAD ILLUSTRATED BY SECTIONS OF HUMAN FETAL MATERIAL

EDMUND APPLEBAUM, D.D.S., AND JOSEPH A. CUTTITA, A.B., M.S., D.D.S

A SHORT course in development of the head is being given to freshmen dental students at Columbia University. Serial sections of human fetal heads are being used to illustrate the anatomical basis of normal growth. This stage of prenatal growth of the head has many advantages for study. At approximately 4 months in utero, the whole head is only the size of a walnut, and the relationship of vessels and nerves to tooth germs and jawbones may be seen in one small slide under low magnification. The higher powers of the microscope can be used to study fine details of these structures.

Each student has several sections of each fetal head so as to obtain a picture of different space levels in a given plane of section. Horizontal, frontal, and sagittal sections of fetal heads at different age levels are also studied, thus giving the student a composite mental picture of the normally growing head as a spacetime structure.

One or two sessions are devoted to orientation, wherein, under low power, the student sees the different sections in broad outline. Then the remaining periods are devoted to more detailed (high-power) study of such subjects as mitosis of cells in enamel epithelium; growth of tooth germs in the fundus; cellular mechanism of enamel hypoplasia; mechanism of bone growth by apposition and resorption; sutural growth; growth of cartilage in the ramus, muscles, blood vessels, and nerves; temporomandibular articulation; Meckel's cartilage; and other structures. In this way the student obtains a dynamic picture of the normal growth of anatomic structures in the head and their mutual relationship.

Obviously a knowledge of anatomy must be accurate before correct deductions as to function and pathology are possible. The low-power pictures presented in these sections are almost like schematic drawings of planes through the head and can be used as a basis for discussing the various interpretations of structure and function in the medical and dental literature.

Fig. 1*A* is a low-power photomicrograph of a typical horizontal section of a human fetal head cut along the plane of the maxillary deciduous tooth germs. Included in the plane of this section, in an anteroposterior direction, are the

Department of Anatomy, School of Dental and Oral Surgery, Columbia University.

germs of the maxillary deciduous teeth, dental lamina, developing maxilla, palate, and cruciate suture. We also see the nasopharynx, body and wings of developing sphenoid bone, cochlea and its cartilaginous capsule, basilar artery, temporal lobe of cerebrum, midbrain, aqueduct of Sylvius, and transverse sinus. Other structures to be noted are the zygomaticus, temporalis, and its attachment to the tip of the coronoid process, some fibers of the masseter and its attachment to the zygomatic bone, the infratemporal fossa, anterior facial vein, and internal carotid artery.

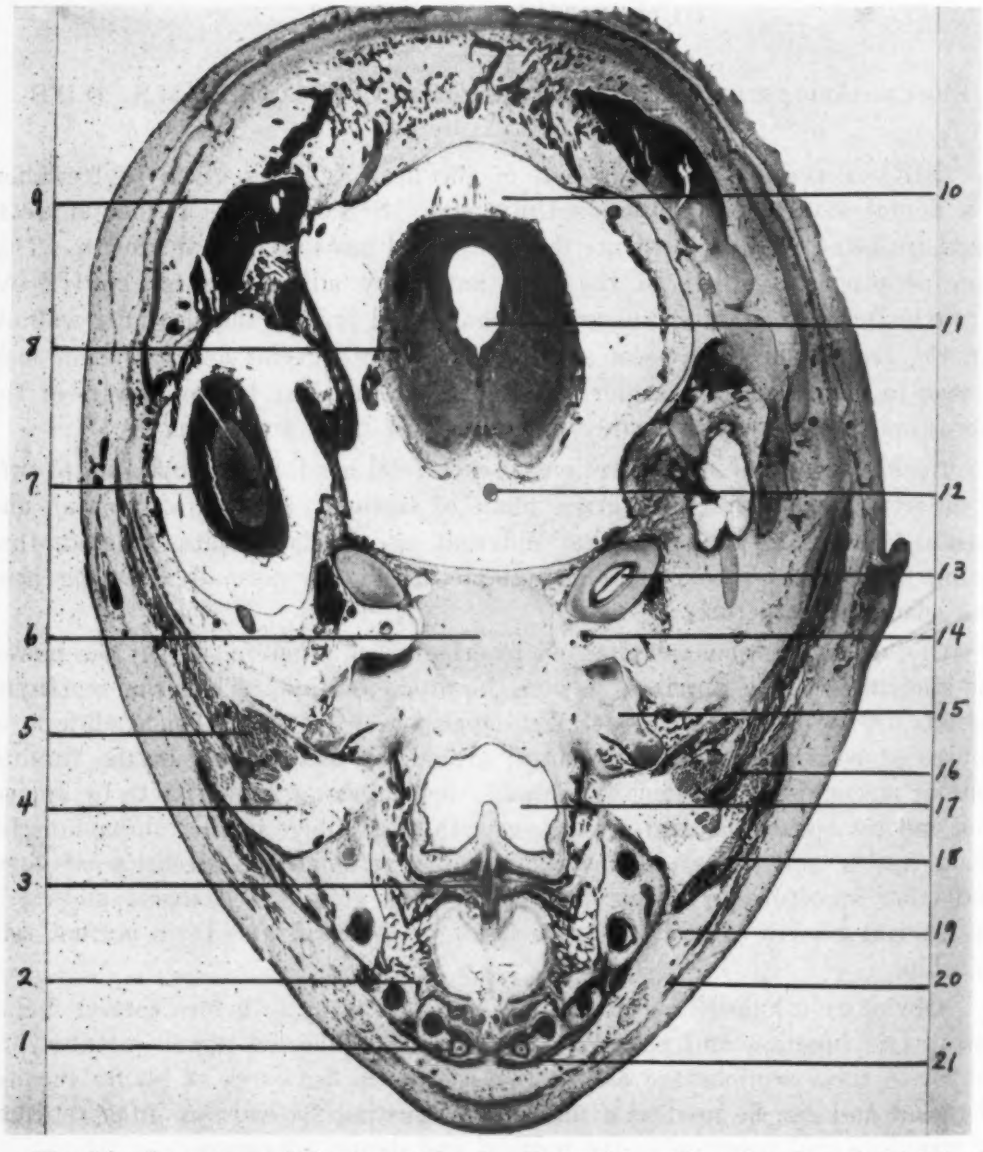


Fig. 1*A*.—Low-power photomicrograph of a horizontal section of a human fetal head. Included in the plane of this section are the following structures: *1*, Maxillary deciduous tooth germ. *2*, Developing maxilla. *3*, Developing palate and cruciate suture. *4*, Infratemporal fossa. *5*, Temporalis. *6*, Body of developing sphenoid. *7*, Temporal lobe of cerebrum. *8*, Midbrain. *9*, Transverse sinus. *10*, Tectum. *11*, Aqueduct of Sylvius. *12*, Basilar artery. *13*, Cochlea and cartilaginous capsule. *14*, Internal carotid artery. *15*, Wing of developing sphenoid. *16*, Attachment of temporalis to coronoid process. *17*, Nasopharynx. *18*, Masseter and attachment to zygoma. *19*, Anterior facial vein. *20*, Zygomaticus. *21*, Lip furrow groove.

Under low power this section is used for orientation. A high-power study can be used to illustrate the growth pattern of the head, especially the mechanism of bone formation. Thus Fig. 1*B* is a high-power view of the cruciate suture seen in Fig. 1*A*. Here we see developing bone trabeculae with osteoid tissue and osteoblasts. This phenomenon is also seen in sections such as Fig. 7*A* showing bone growth at the symphysis, maxillozygomatic and palatal sutures. When these are studied in conjunction with sections such as Fig. 4 showing resorption of bone around the expanding tooth germ, the student gains an insight into the mechanism of bone modeling, an important problem in the study of orthodontics.[4-6]

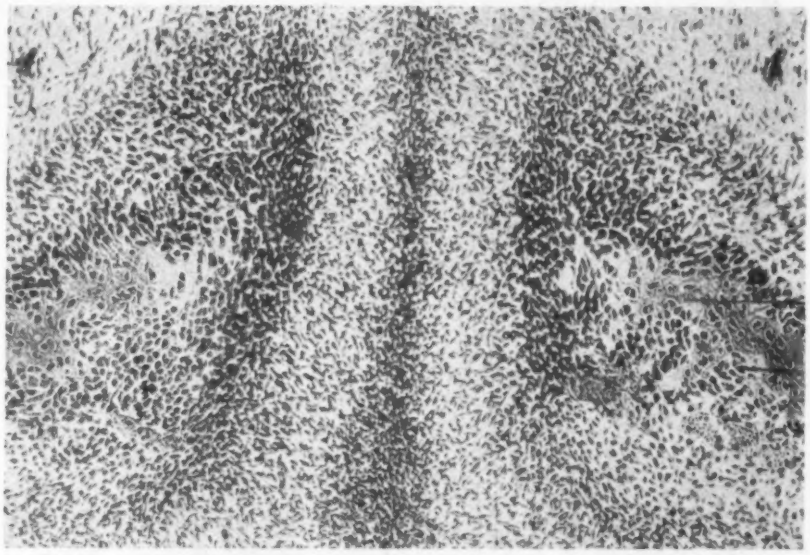


Fig. 1*B*.—Photomicrograph of cruciate suture seen in Fig. 1*A* (magnification ×200). *A*, Developing bone trabecula. *B*, Osteoblasts.

Fig. 2 is a low-power photomicrograph of a sagittal section of a human fetal head. Included in the plane of this section in an anteroposterior direction are the lips, enamel organs of maxillary and mandibular deciduous incisors, the developing maxilla, palate, and mandible, muscles of the tongue and floor of the mouth, hyoid bone, epiglottis, and pharynx. We may also note the odontoid process of the second cervical vertebra, the pons, sphenoid bone and pituitary body, nasal bones and conchae.

A low-power study of this section can be used to illustrate the free airway to be maintained during general anesthesia and the danger of forcing throat packs back too far in oral surgery procedures. Under high-power (oil immersion) we can see mitotic figures in the growing enamel epithelium of the tooth germs (Fig. 3).

Fig. 4 is a low-power photomicrograph of another sagittal section through the midline of a slightly older human fetus. The enamel organ is beginning to function in the production of enamel and dentine.[1] Growth of the dental follicle causes resorption of the surrounding bone, enlarging the area for growth

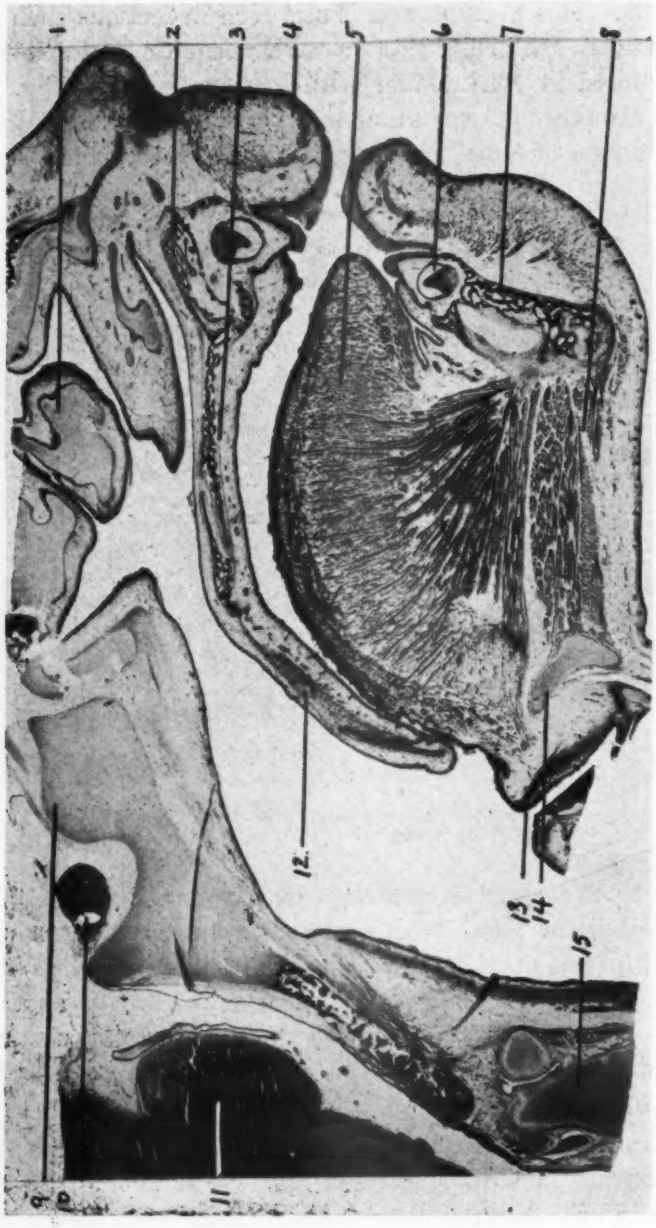


Fig. 2.—Photomicrograph of a sagittal section of human fetal head. *1*, Nasal bones. *2*, Maxilla. *3*, Palate. *4*, Lips. *5*, Tongue. *6*, Enamel organ of mandibular deciduous central incisor. *7*, Mandible. *8*, Muscles of the floor of the mouth. *9*, Sphenoid bone. *10*, Pituitary body. *11*, Pons. *12*, Soft palate. *13*, Epiglottis. *14*, Hyoid bone. *15*, Odontoid process of second cervical vertebra.

of the crown germ.[2] This can be seen under higher power. At the same time there is apposition of new bone on the outer surface of the mandible and maxilla.

In this section we see that the cervical loop of the enamel organ grows deeper into the jawbone, as evidenced by resorption of bone in the fundus. Only on completion of the crown of the tooth and with beginning of root formation does Hertwig's epithelial sheath appear and remain a relatively fixed point during growth of the root and eruption of the tooth.[3] This point is demonstrated by sections of older specimens.

Fig. 5 is an oil-immersion study of the enamel formation of one of the tooth germs seen in Fig. 4. Here we note cystic areas in the ameloblasts, and this presents an interesting topic for discussion, namely, the mechanism of enamel hypoplasia resulting from a disturbance of the ameloblasts.[1]

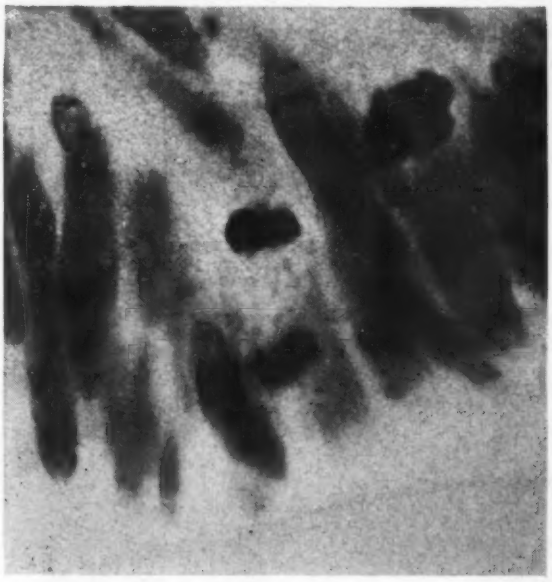


Fig. 3.—Oil-immersion photomicrograph of mitotic figures in growing enamel epithelium of tooth germ seen in Fig. 2.

Fig. 6 is a low-power photomicrograph of a sagittal section, cut lateral to the midline along the body and ramus of the mandible. This section shows the developing ramus of the mandible with its cartilaginous bone formation at the condyle (similar to development of long bones); apposition of bone at the posterior border and resorption of bone at the anterior border of the ramus; the developing tooth germs with bone of the body of the mandible below, while as yet there is no bone at the crest. It is noteworthy that so little bone is present in body of mandible at this stage of development.[5] We also see the attachment of the temporal muscle at the developing coronoid process, the insertion of the masseter at the angle of the mandible, the platysma and some fibers of the external muscles of expression, the anterior facial artery and vein, and a part of the parotid gland with its related blood vessels and nerves (branches of external carotid artery and the temporal and internal maxillary

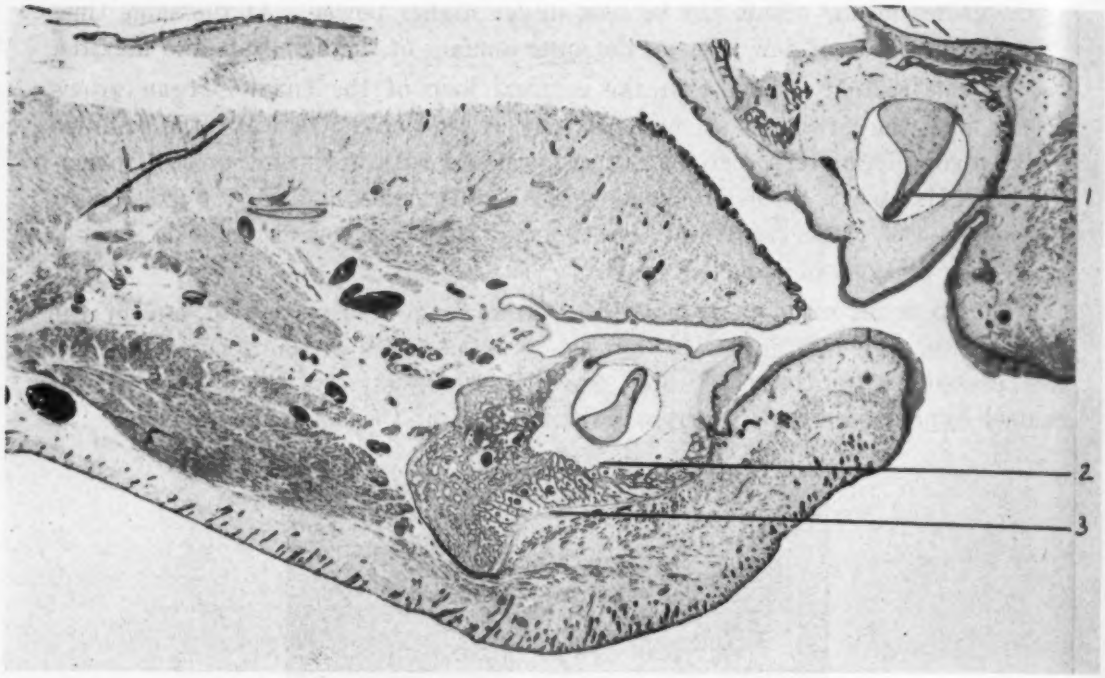


Fig. 4.—Photomicrograph of sagittal section through midline of slightly older fetus. *1*, Beginning formation of enamel and dentine. *2*, Resorption of bone in fundus of crypt of expanding tooth germ. *3*, Apposition of bone on surface of mandible.

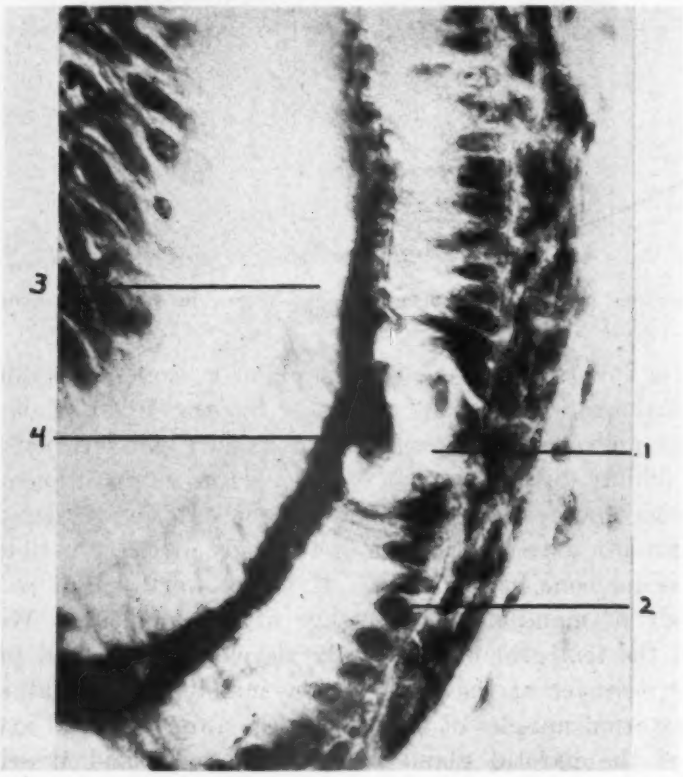


Fig. 5.—High-power photomicrograph of an area of cystic degeneration in enamel organ of Fig. 4. *1*, Cystic area. *2*, Ameloblasts. *3*, Forming dentine. *4*, Forming enamel.

veins, facial and great auricular nerves). In this section the two different types of bone growth in the mandible can also be demonstrated to the student.

Fig. 7*A* is a frontal section through the symphysis of the mandible and the anterior portion of the eyeball and nose. This is an excellent illustration of bone growth at the symphysis, suture of hard palate, and the maxillozygomatic suture. The infraorbital canal with its blood vessels and nerves is seen below the inferior rectus muscle of the eye. The nasal bones and conchae, the buccinator muscle, some fibers of the platysma and other muscles of expression, and several tooth germs are presented for study.

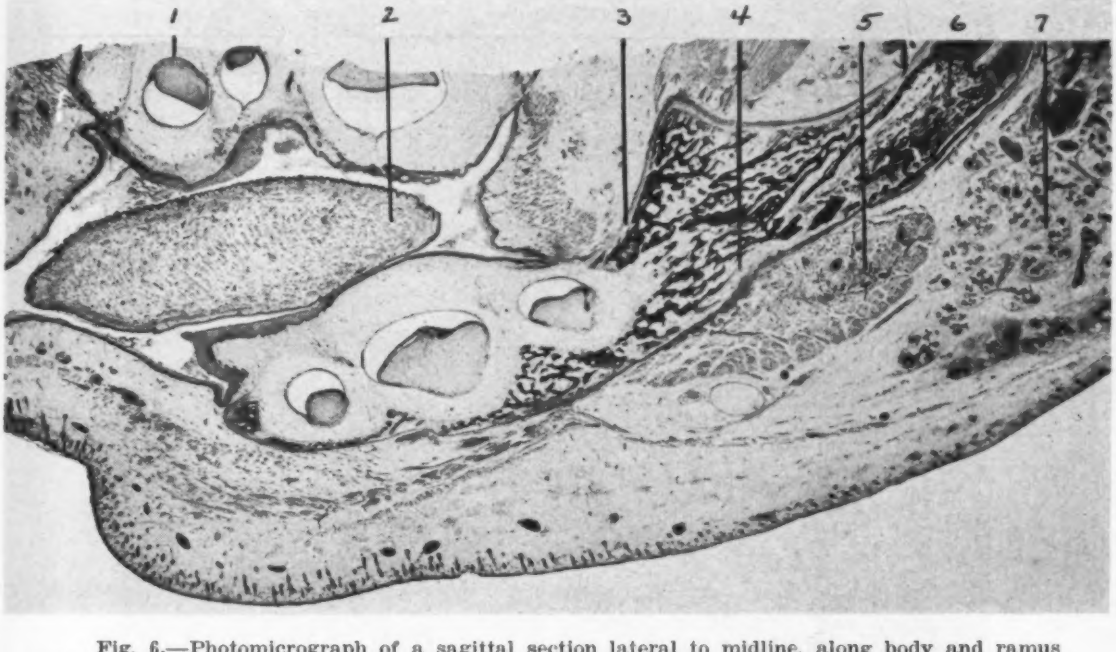


Fig. 6.—Photomicrograph of a sagittal section lateral to midline, along body and ramus of the mandible. *1*, Tooth germ. *2*, Tongue. *3*, Resorption of bone. *4*, Apposition of bone. *5*, Masseter muscle. *6*, Cartilaginous bone formation. *7*, Parotid gland.

Fig. 7*B* is also a frontal section but in a plane more posterior to Fig. 7*A*. The muscles of the floor of the mouth stand out in this section. From below upward we see the platysma, anterior belly of the digastric, the mylohyoid and its insertion in the mylohyoid groove on the lingual surface of the mandible (above Meckel's cartilage in this section). Next in order is seen the geniohyoid, genioglossus, some fibers of hyoglossus lateral to genioglossus and the intrinsic muscles of the tongue. Between hyoglossus and mylohyoid the hypoglossal nerve can be found. We may also see the internal and external rectus muscles, the inferior rectus and inferior oblique muscles of the eye, the optic nerve and infraorbital nerve and vessels, the temporalis and masseter muscles and a section of the zygomatic bone.

The student identifies the muscles of the floor of the mouth, noting the relation of one muscle to another and of each muscle of the group to surrounding structures. In some sections blood vessels and nerves are found in or around the muscles. Wherever possible these are identified and their origin and

course is studied. The importance of this group of muscles in dentistry is brought out by a discussion of the paths through which dentoalveolar abscesses and infections may spread along the fascial planes of these muscles into the neck regions. In these conditions a thorough knowledge of the anatomical structures and their relationships is essential to the oral surgeon.

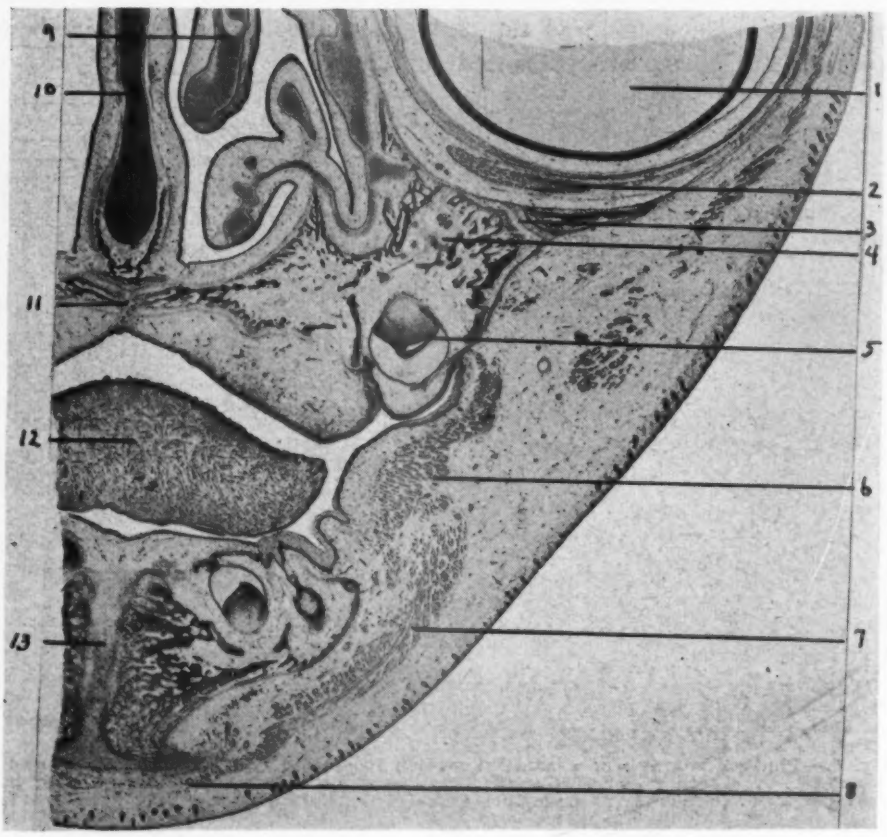


Fig. 7A.—Photomicrograph of a frontal section of a human fetal head. *1*, Eyeball. *2*, Inferior rectus. *3*, Maxillozygomatic suture. *4*, Infraorbital canal. *5*, Maxillary tooth germ. *6*, Buccinator. 7, External muscles of expression. *8*, Platysma. *9*, Nasal conchae. *10*, Nasal septum. *11*, Palatal suture. *12*, Tongue. *13*, Symphysis of mandible.

Fig. 8 is a horizontal section through the developing temporomandibular joint. Here we see the meniscus interposed between the head of the condyle and the glenoid fossa of the temporal bone. The external pterygoid muscle is attached to the head of the condyle and it can be followed to its origin on the outer surface of the lateral pterygoid plate. The section cuts through the uppermost part of the ramus and we can see the attachment of the masseter muscle on its external surface and some fibers of the temporalis on the inner surface of the coronoid process. Posterior to the temporomandibular joint we see the head of the malleus, one of the bones of the middle ear, while mesial to this we have a section of the cochlea with its cartilaginous capsule. Anterior to the cochlea the internal carotid artery may be noted while posterior to it, the seventh nerve and the vestibular division of the eighth have just entered from the brain cavity.

Mesial to the lateral pterygoid plate the tensor veli palatini can be seen with its tendon winding around the hamular process, and further mesially we note the Eustachian tube branching out from the nasopharynx. Posterior to the external pterygoid muscle we note a group of blood vessels, branches of the internal maxillary artery, and a large nerve trunk which appears to be the mandibular.

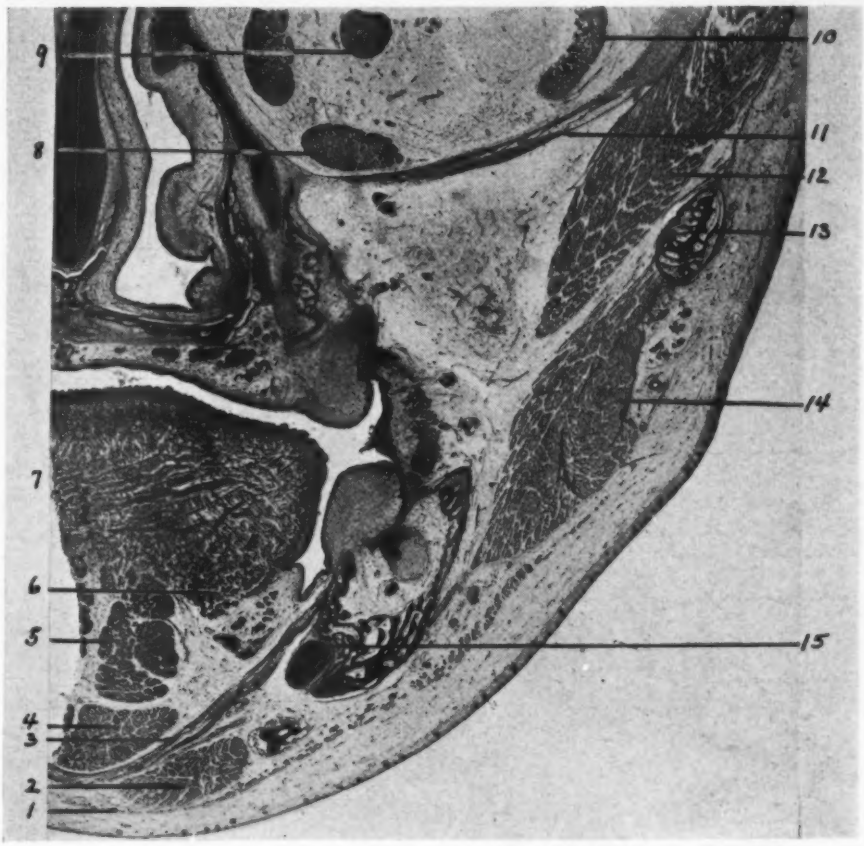


Fig. 7*B*.—Photomicrograph of another frontal section cut in plane posterior to Fig. 7*A*. *1*, Platysma. *2*, Anterior belly of digastric. *3*, Mylohyoid. *4*, Geniohyoid. *5*, Genioglossus. *6*, Hyoglossus. *7*, Tongue. *8*, Inferior rectus. *9*, Optic nerve. *10*, External rectus. *11*, Inferior oblique. *12*, Temporalis. *13*, Zygoma. *14*, Masseter. *15*, Meckel's cartilage.

A study of this section will point out to the student the close relationship between the temporomandibular joint, the muscles of mastication, and the middle and internal ear structures, and will offer a basis for discussion of the literature on the relationship between hearing and disturbances in the temporomandibular joint, which may be associated with closed bite.

Fig. 9 is a photomicrograph of a section of a human fetal head cut through the body of the mandible and Meckel's cartilage. This section is used for the study of Meckel's cartilage and its relationship to the mandible and surrounding structures at this stage of development. Posteriorly we see this structure lying adjacent and lingual to the body of the mandible. As we follow it anteriorly, it dips downward and then turns laterally to be incorporated into the body of

the mandible. In the region near the symphysis (in this section) we see bone growth in what appears to have been the former outline of Meckel's cartilage. In addition, this section presents for study the masseter, internal pterygoid, platysma, mylohyoid, hyoglossus, and genioglossus muscles, parts of the submaxillary and sublingual glands and Wharton's duct, the hypoglossal and lingual nerves, and the mandibular suture.

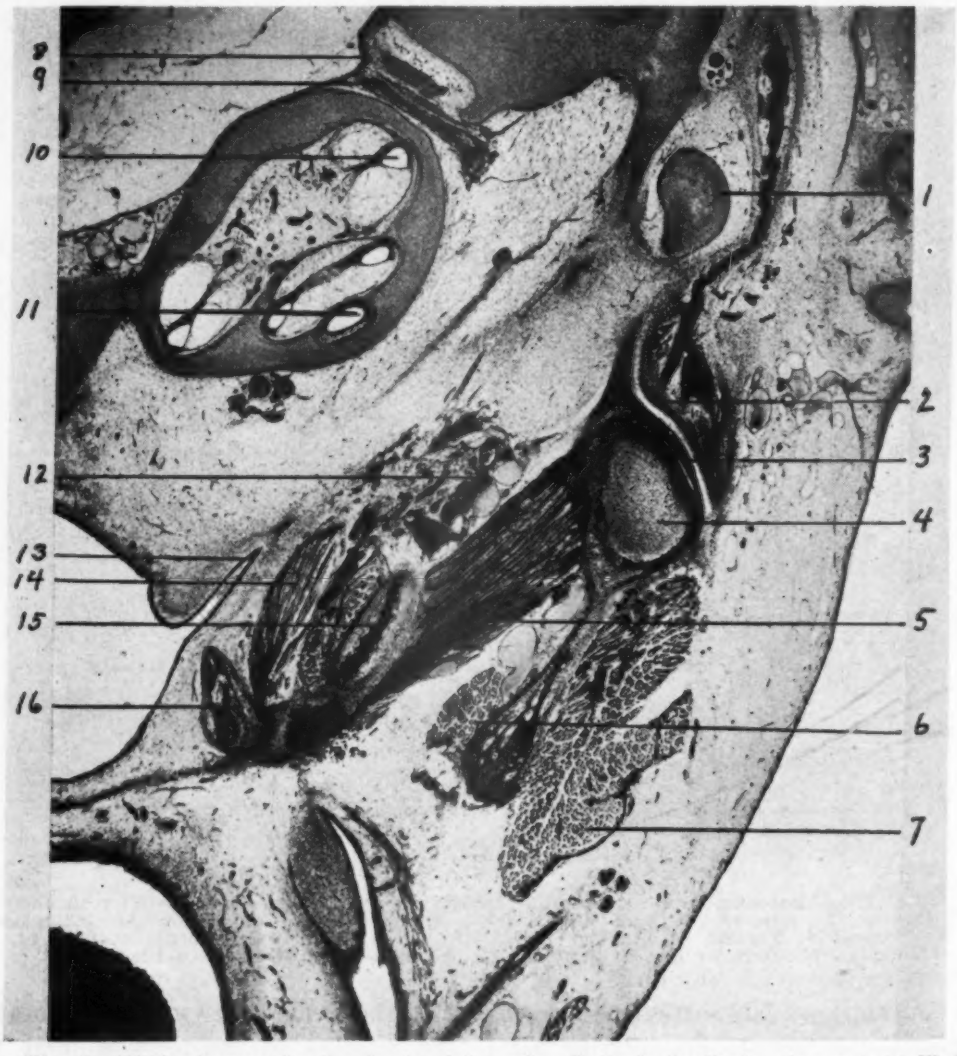


Fig. 8.—Photomicrograph of a horizontal section through developing temporomandibular joint. *1*, Malleus. *2*, Temporal bone (glenoid fossa). *3*, Meniscus. *4*, Head of condyle. *5*, External pterygoid. *6*, Coronoid process and temperalis. *7*, Masseter. *8*, Vestibular division of eighth nerve. *9*, Seventh nerve. *10*, Ductus cochlearis. *11*, Helicotrema. *12*, Mandibular nerve. *13*, Eus achian tube. *14*, Tensor veli palatini. *15*, Lateral pterygoid plate. *16*, Hamular process.

The object of the course is to acquaint the student with the fact that dentistry cannot be concerned only with the teeth, but that a knowledge of the anatomy and physiology of their supporting structures and related tissues is essential in orthodontics, oral surgery, prosthetics, etc. The dentist who sees nothing but teeth can at best be only a good mechanic. The student who

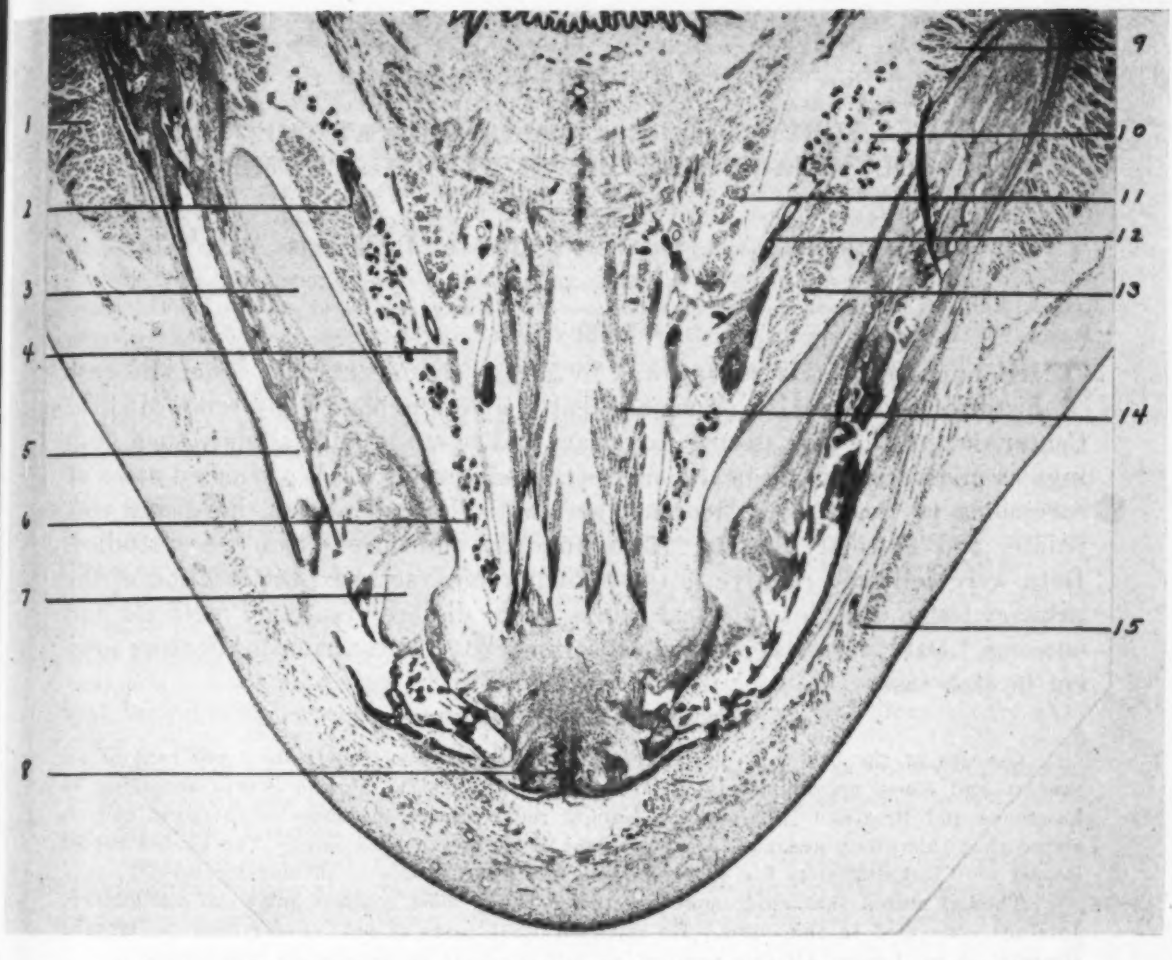


Fig. 9.—Photomicrograph of a horizontal section through the body of the mandible and Meckel's cartilage. *1*, Masseter. *2*, Hypoglossal nerve. *3*, Meckel's cartilage. *4*, Lingual nerve. *5*, Body of mandible. *6*, Sublingual gland. *7*, Meckel's cartilage. *8*, Symphysis. *9*, Internal pterygoid. *10*, Submaxillary gland. *11*, Hyoglossus. *12*, Wharton's duct. *13*, Mylohyoid. *14*, Genioglossus. *15*, Platysma.

appreciates the dynamic function of the mandible and maxilla in terms of muscle attachment and muscle action, bone growth and resorption, blood and nerve supply, and the relationship of all these structures to the dental apparatus as a whole has an essential and invaluable background for the rational practice of dentistry.

The authors wish to acknowledge the cooperation of Dr. Moses Diamond and Dr. William Rogers of the anatomy department in the organization and preparation of this course.

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A SURVEY OF ORAL CARCINOMA AT THE COLUMBIA-PRESBYTERIAN MEDICAL CENTER

WILLIAM H. ARCHER, A.B., D.D.S., AND ROBERT D. MORRIS, A.B., D.D.S.

INTRODUCTION

THE purpose of this survey was to gather significant data from the case histories of the oral carcinomas treated at Columbia-Presbyterian Medical Center during the past twenty-five years and to study and tabulate such findings as might contribute to the oral carcinoma problem. We selected cases of carcinoma of the tongue, floor of the mouth, buccal mucosa, hard and soft palate, and alveolar gingiva. Two hundred and three cases were studied. Data were collected relative to the patient's age, race, sex, the location of the primary lesion, the chronological course of the disease, treatment methods, and outcome. Data were also secured concerning possible contributing factors present in each case.

REVIEW OF THE LITERATURE

Cancers of the oral and pharyngeal mucous membranes constitute 3 per cent of all cancers and cause approximately 3,700 deaths yearly in the United States according to Lawrence and Brezina.[1] A Canadian group,[2] reporting on 464 cases of intraoral cancer, stated that this group accounts for 5 per cent of all new cancer cases. The United States Report on Vital Statistics for 1939 recorded 5,012 deaths due to intraoral cancer.[24]

Thoma[3] stated that epidermoid carcinoma is the most common intraoral malignancy. Jorstad[4] concurred in this view. He reported 1,561 cases of oral cancer seen at Barnard Hospital in St. Louis. Of this number, all but ten were squamous-cell carcinomas.

Oral cancer occurs principally in the cheek, alveolar gingiva, palate, tongue, and the floor of the mouth.[1] Liljencrantz,[5] reporting on autopsy findings, in 1,000 consecutive cases of cancer, stated that 60 per cent of intraoral cancer occurred in the tongue, 20 per cent in the floor of the mouth, 10 per cent in the cheeks, and 5 per cent each in the gingiva and palate. Keyes,[6] reporting on 736 cases, found 37 per cent occurred in the buccal mucosa (all mucous membrane except the tongue), while 11 per cent involved the tongue. Keyes,[7] in another report, stated that of 406 mouth cancers, 28.6 per cent involved the buccal mucosa and 40.9 per cent the tongue. Padgett[8] reported 43 per cent of all mouth cancers to be in the tongue, anterior to the circumvallate papillae, and 10 per cent in the floor of the mouth.

Sex.—Oral carcinoma occurs more often in men than in women according to Liljencrantz.[5] Eighty-five per cent of 464 patients were men in one study.[2] In another, Jorstad[4] reported a ratio of 6 men to 1 woman, and Richards[9] reported 10 men to 1 woman in his study of 55 cases. Welsh and Nathanson[10] stated that of 835 oral cancers, 92.6 per cent were men and 7.4 per cent were women. Burnham[11] reported 165 cases of which 128 cases, or 77.5 per cent, were men, and 37 cases, or 22.5 per cent, were women. This is the highest incidence in women reported.

Age.—Oral cancer is an affliction of advancing age. Padgett[8] found that most cases occurred between 50 and 60 years of age. After the sixth decade the actual number de-

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creased, but the percentage increased. Jorstad[4] found that the age of those so affected ranged from 35 to 83 years with the average age between 62 and 65 years. A Canadian group[2] stated that approximately one-half of cancers of the mouth occur between 60 and 69 with the other half occurring after 70 years of age. In females, however, 50 per cent of oral carcinomas were discovered prior to the age of 50 years.

Possible Contributing Factors to Etiology.—Those conditions which produce or prolong a chronic inflammatory reaction are mostly associated with some increase in the incidence of cancer in the oral region according to Padgett.[8] He reported that 75 per cent of all epidermoid carcinomas of the oral cavity are preceded by some extrinsic factor.

Syphilis is often cited as the outstanding example of predisposition to cancer. Kaplan[12] stated that syphilis is a concomitant disease in 40 to 50 per cent of all oral cancer. Martin[13] found that one-third of all tongue cancer patients had syphilis. Fraser[14] placed the incidence of syphilis in cases of carcinoma of the tongue as a whole at 42.3 per cent and in carcinoma of the dorsum of the tongue at 78.3 per cent. Lund[15] stated that the relationship of syphilis to cancer is most important in cancer of the tongue where the association exists in between 17 and 32 per cent of all cases. However, in all other locations, the presence of a positive history of syphilis or of a positive Wassermann test is so low that the incidence may be assumed to be close to the normal in the general population.[15]

Leukoplakia is also often cited as a common precancerous lesion. Fournier[16] and von Bergman[17] estimated that leukoplakia preceded 30 per cent of the cancers of the oral cavity. Fraser[14] reported 90 per cent of carcinomas of the dorsum of the tongue to be preceded by leukoplakia. Sturges and Lund[25] reported 298 patients who had leukoplakia with no malignancy at the time of original examination. Twelve of these patients, however, developed cancer later. They stated that according to their calculations, patients with leukoplakia developed oral cancer twelve times more frequently than chance alone would predict.

Blair and Byars[18] stated that leukoplakia may be the result of smoking or chewing tobacco. Lamb and Eastland[19] found that the use of tobacco predisposed to leukoplakia and finally to carcinoma.

According to Grace[20] cancer of the mouth, lips, tongue, larynx, and pharynx is more prevalent in smokers than in nonsmokers, and is most frequently seen in pipe and cigar smokers who rarely inhale and in whom the irritating substance, tar, is locally deposited.

Survival.—Lawrence and Brezina[1] reported their best five-year survival rates in patients with cancer of the anterior tongue and buccal mucosa. Welsh and Nathanson[10] stated that life expectancy is better for carcinoma of the buccal mucosa than for any other type of intraoral cancer due to the accessibility for treatment of the lesion and the fact that the tumors in that area were usually well differentiated and frequently of a papillary type. They also stated that life expectancy is slightly greater in the female than in the male. Richards[9] concurred in the belief that carcinomas of the buccal mucosa and tongue are treated more successfully than lesions of other locations in the oral cavity.

Five-year survival rates for oral carcinoma are not high. Patterson[21] reported 32 per cent in a series of 602 cases treated by radiation. Pfahler[22] reported a similar study of 438 cases (treated by radiation) with a 29 per cent five-year cure obtained.

Martin and Pfleuger[23] reported a five-year survival period among 30 per cent of 99 unselected cases at Memorial Hospital of New York City. Welsh and Nathanson[10] summarized their findings in 2,345 cases as follows: The median life expectancy of untreated cases was ten months, while in treated cases it was seventeen months in males and twenty-one months in females. The authors felt that age did not influence life expectancy but, when compared with normal life expectancy, carcinoma was most malignant in the young.

OUR STUDY

Experimental Group.—Two hundred and three case histories of oral carcinoma treated at Columbia-Presbyterian Medical Center and its constituent hospitals in the past twenty-five years were examined. The lesions were re-

corded as being found in the following locations: tongue, floor of the mouth, cheek, palate, and alveolar gingiva.

We recorded the age, sex, race, and occupation of the patient. Certain etiological factors were tabulated: syphilis, leukoplakia, habits (especially smoking), and the possibility of irritation (badly broken-down natural teeth or artificial restorations). We also recorded the location of primary lesions, time of onset, date of diagnosis, symptoms, the type of treatment, and the outcome of the treatment.

Control Group.—In order to aid in the evaluation of our findings, 429 case histories of patients in the same age range (above 35 years of age) were selected at random from the records at Columbia-Presbyterian Medical Center. These constituted a control group. Sex, race, and incidence of syphilis were noted. These patients presented with various conditions other than carcinoma such as pregnancy, infection diseases, disturbances of vision, etc.

Diagnosis.—Biopsy disclosed that 199 cases or 98 per cent of the oral carcinomas were the squamous-cell type, 3 were cylindromatous carcinomas, and one was an adenocarcinoma.

Age.—The patients, at the time of diagnosis, ranged in age from 26 to 84 years. Only four cases occurred in individuals under 35 years and one case was in a person over 80 years. Seventy-eight percent of the cases fell in the 45- to 74-year age range.

Age in years	35-44	45-54	55-64	65-74	75-84
Number of cases	18	58	57	53	13
Per cent of total	8.9	28.6	28.1	26.1	6.4

Sex.—Eighty-five per cent of our cases were males; 15 per cent were females. In our control group 42.4 per cent of the patients were males and 57.6 per cent were females. Our sex-carcinoma ratio is in agreement with the literature cited in the general discussion.[2, 4, 5, 7, 9-11]

Race.—One hundred and eighty-six cases were of known nationality or race. If the patient stated that both his parents were of the same foreign ethnic group, he was considered in that group regardless of his birthplace. The predominant groups were:

	ORAL CARCINOMA GROUP (PER CENT)	CONTROL GROUP (PER CENT)
Negro	4.8	16.8
White American	37.1	
Irish	21.5	12.6
German	9.7	

The remaining 27 per cent of the oral carcinoma group were distributed among 15 smaller ethnic groups.

A significant variation of the oral carcinoma group from the control group was observed in only the Negro and Irish segments. These figures would seem to indicate a relative predisposition in those of Irish extraction and a lessened tendency in Negroes.

Location.—The location of the primary lesion was taken as a basis for subdivision. The incidence of carcinoma in the various locations was:

	NUMBER OF CASES	FIVE-YEAR SURVIVALS (PER CENT)
*Anterior tongue	63	23.8
Tongue and floor of mouth	79	24.0
Buccal mucosa	18	22.2
Alveolar gingiva and palate	36	22.2

There were 7 oral carcinomas of such extension that they involved more than one of the above groups. These 7 cases were not considered under any of the above groups. However, all other information gathered from these was considered under various headings.

Possible Contributing Factors.—Twenty-six cases, or 12.3 per cent of the total, were regarded as syphilitic because of a positive Wassermann test or a definite medical history. Of these 26 cases, 20, or 77 per cent, were associated with carcinoma of the anterior tongue, while 32 per cent of the carcinomas of anterior tongue were associated with syphilis. These findings concur with those of Lund[15] who states that syphilis is important in carcinoma of the tongue but that in all other locations, the incidence of syphilis is so low that it may be assumed to be close to the normal incidence in general population.

Leukoplakia was found to be present in 32 cases or 15.8 per cent, in contrast to the 6 per cent cited by Jorstad[4] and the 30 per cent estimate of Fournier[16] and von Bergman.[17] Of these 32 cases, 11 also had syphilis.

Smoking was considered as a contributing factor. Patients were classified as heavy, moderate, light, or nonsmokers. The first group included constant pipe or cigar smokers and those who smoked in excess of 40 cigarettes per day. The second group, the moderate smokers, were those who smoked several cigars or pipefuls or about 20 cigarettes per day. The third group consisted of those who did not smoke or smoked only occasionally. The results of the tabulation were:

SMOKERS	NUMBER OF CASES	PER CENT OF TOTAL
Heavy	39	19
Moderate	47	23
Light or nonsmokers	117	58

On the basis of these findings smoking was considered to be negligible as an extrinsic predisposing factor. Eighty-one per cent of the total number of patients were classified as moderate, light, or nonsmokers, and 19 per cent as heavy smokers.

Trauma and irritation from faulty natural or artificial teeth have often been mentioned as extrinsic predisposing factors in oral carcinoma. Our cases were classified in groups as follows: The first group comprised those with a history of trauma or possible irritation other than that caused by a denture. These constituted 7 per cent of the total group. The second group consisted of those who wore dentures, and in whom denture irritation was thus possible. Few of these actually had evidence of denture irritation. These made up 5

*Anterior tongue—anterior to circumvallate papillae.
Tongue and floor of mouth—all carcinomas of the tongue and floor of the mouth except those involving only the anterior tongue.
Buccal mucosa—the mucous membrane which lines the cheeks and inner lips from the mucobuccal fold of the lower to the mucobuccal fold of the upper.
Alveolar gingiva—the attached gingiva.
Palate—hard and soft.

per cent of the total group. Thus the number of patients with a history of trauma or possible irritation due to any dental factor was 12 per cent of 203 cases.

It is interesting to note that of the 203 cases at least 116, or 57 per cent, gave no evidence of any one of the possible contributing factors, such as syphilis, leukoplakia, heavy smoking, trauma, and irritation from various sources.

The occupation of each patient was recorded with a view toward etiology. The occupation of carpentry was of particular interest because of the frequently stated theory that the habit of holding nails, screws, etc., in the mouth predisposed to carcinoma. Of our 203 cases, only 3 were carpenters—less than 2 per cent of the total. The other occupations were diversified with no trend apparent, and no significance could be attached to them.

Treatment.—Of the 203 cases recorded, 185 were treated by surgery, radiation, or a combination of the two. The remainder either were not treated at this institution or were given such therapy as cautery or colloidal lead. Twenty-five cases were treated surgically; 101 cases by radiation (x-ray radium or radon seeds); 20 cases were treated with a combination of radiation and surgery.

Of the 185 cases treated, at the time of the last follow-up 51 were still alive, 134 had died. The 134 cases had survived an average of thirty-eight months from onset until death. Twenty-four, or 18 per cent, survived over five years. Of the 51 patients still alive, 23 had been under observation for over five years. Thus a total of 47, or 25 per cent of the group of 185, survived at least five years. It should be noted that this percentage might be increased slightly with subsequent five-year examinations in that group which had been under observation for less than five years.

TREATMENT RESULTS

	SURGERY	RADIATION	RADIATION AND SURGERY	TOTAL
Total patients treated	25	140	20	185
Total recorded deaths	15	101	18	134
Average time survived (months)	52	33	56	38
Number who survived 5 years or more	5—33%	13—13%	6—33%	24—18%
Number still living	10	39	2	51
Number of 5-year survivals among living	6	16	1	23
Total of 5-year survivals in both groups	11—44%	29—21%	7—35%	47—25%

Of the 185 cases treated, 135 were diagnosed before September, 1940, allowing five years for follow-up. Of these 135 cases, 38, or 28 per cent, were five-year survivals, all the remaining having died within five years. For the entire group of 203 cases, the five-year survival rate was 25 per cent, but this might be increased by subsequent five-year examinations. The 28 per cent five-year survivals in our group were in close agreement with the 30 per cent of Martin and Pfleuger,[23] the 29 per cent of Pfahler,[22] the 32 per cent reported by Patterson.[21]

SUMMARY AND CONCLUSIONS

1. Two hundred and three case histories of oral carcinoma were studied statistically. It was found that carcinoma of the mouth was essentially a

disease of middle age. Seventy-eight per cent of the cases occurred in patients between 45 and 74 years of age.

2. Eighty-five per cent of patients were males.

3. A study of the ethnic distribution disclosed higher incidence of oral carcinoma in persons of Irish extraction and a lower incidence in Negroes.

4. The possible relationship of syphilis as a contributing factor was indicated only in carcinoma of the anterior tongue. Thirty-two per cent of such patients had syphilis. Seventy-seven per cent of all syphilis cases in our group were associated with anterior tongue carcinomas. Syphilis did not appear significant in other locations.

5. Trauma and irritation from broken teeth, artificial dentures, smoking, etc., as extrinsic contributing factors were found to be insignificant.

6. Survival, based on a five-year period, bears no direct relationship to location of the primary lesion. Comparable five-year survival rates were obtained in all locations studied. They were: anterior tongue, 23.8 per cent; tongue and floor of the mouth, 24 per cent; buccal mucosa, 22.2 per cent; alveolar gingiva and palate, 22.2 per cent.

7. Of the 135 patients who were followed for a five-year period, 28 per cent survived.

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THE EFFECT OF ZINC OXIDE EUGENOL CEMENT ON PENICILLIN

HENRY A. BARTELS, B.S., D.D.S.*

SHAW, Sprawson, and May, in a recent article[1] on the efficacy of penicillin in root canal therapy, wrote, "Zinc oxide with eugenol was observed to destroy penicillin completely" (page 552, paragraph 2, line 7). They therefore advocated sealing in the penicillin-saturated absorbent point with a layer of wax, followed by zinc oxyphosphate cement. No evidence, however, was submitted by them to substantiate their statement. Since their and our experience did not coincide, the following tests were performed to obtain further data on this subject.

PROCEDURE

Small flasks of infusion agar were cooled to 45° C. and inoculated with a fresh suspension of a penicillin-sensitive strain of *Staphylococcus aureus*. The medium was poured into plates and allowed to solidify. Mixtures of zinc oxide and eugenol alone and together with various concentrations of penicillin in physiologic salt solution were placed directly on the surface of the medium. Liquids such as penicillin in solution, eugenol, or combinations of both were pipetted into cuplike depressions made in the agar by means of a sterile cork borer. The walls and floor of these "cups" were rimmed with a small amount of cooled liquid agar to seal any minute cracks produced by use of the cork borer. The penicillin was tested in varied unit strength. Since eugenol alone or when mixed with zinc oxide possesses a definite bacteriostatic activity, it was necessary to test these materials separately as well as in combination with penicillin. The various units of penicillin used were incorporated with the zinc oxide and eugenol by thorough spatulation. The more concentrated penicillin dilutions caused the zinc oxide eugenol to become light yellowish in color. The plates were incubated twenty-four hours at 37° C., and the clear zones about the test materials were measured with a millimeter rule. The size of these areas in which no growth was observed indicated the inhibitory activity of the various substances tested. (Table I, Figs. 1 and 2.)

The results of Table I indicate that:

1. Zinc oxide, dry or moist as a paste, exerts little if any bacteriostatic activity when tested by this method. Possibly in a liquid medium where ionization could occur, the zinc oxide would be shown to possess some inhibitory quality.

2. Eugenol, alone or when mixed with zinc oxide, has a definite inhibitory activity, preventing growth in a zone of 6 mm. radius.

3. Eugenol, alone or mixed with zinc oxide, does have an effect on the potency of penicillin concentrations. In the higher dilutions, 0.4 to 40 units, this effect is more noticeable than in the more concentrated dilutions (200 and 2,000 units). In the former dilutions when combined with zinc oxide and eugenol, the drop in potency is sufficient to reduce the activity of the peni-

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TABLE I. INHIBITION OF STAPHYLOCOCCUS AUREUS BY TEST MATERIALS

TEST MATERIAL	UNITS	INHIBITION ZONE IN MILLIMETERS*
Penicillin sodium	0.4	9
Penicillin sodium	20	15
Penicillin sodium	40	18
Penicillin sodium	200	21
Penicillin sodium	2,000	23
Penicillin calcium	2,000	22
Zinc oxide powder dry		0
Zinc oxide powder moist		0
Zinc oxide + penicillin sodium	0.4	8
Zinc oxide + penicillin sodium	20	12
Zinc oxide + penicillin sodium	40	17
Zinc oxide + penicillin sodium	200	20
Zinc oxide + penicillin sodium	2,000	21
Zinc oxide + penicillin calcium	2,000	19
Eugenol		6
Eugenol + penicillin sodium	0.4	5
Eugenol + penicillin sodium	20	7
Eugenol + penicillin sodium	40	10
Eugenol + penicillin sodium	200	17
Eugenol + penicillin sodium	2,000	19
Eugenol + penicillin calcium	2,000	19
Zinc oxide and Eugenol		6
Zinc oxide, eugenol, + penicillin sodium	0.4	5-6
Zinc oxide, eugenol, + penicillin sodium	20	8
Zinc oxide, eugenol, + penicillin sodium	40	9
Zinc oxide, eugenol, + penicillin sodium	200	16
Zinc oxide, eugenol, + penicillin sodium	2,000	18
Zinc oxide, eugenol, + penicillin calcium	2,000	19

*Average of three tests.

cillin to the range of inhibition of the zinc oxide and eugenol mixture acting alone. In the more concentrated dilutions of penicillin, sufficient potency is still present to produce areas of inhibition of bacterial growth definitely larger than are caused by zinc oxide and eugenol.

Further tests were performed by saturating absorbent points with various unit concentrations of penicillin, and placing them upon the surface of an agar medium previously inoculated as described above with *Staphylococcus aureus.* In some instances zinc oxide and eugenol were brought directly in contact with the larger end of the penicillin-saturated absorbent points; in other instances, mineral oil was placed at the end of the points so that the zinc oxide-eugenol paste, when added, would not come in direct contact with the end of the absorbent point carrying the penicillin. (Table II, Figs. 3 and 4.)

TABLE II. THE EFFECT OF ZINC OXIDE-EUGENOL ON PENICILLIN CARRIED ON ABSORBENT POINTS

		PENICILLIN POINTS IN CONTACT WITH		
			ZINC OXIDE EUGENOL	MINERAL OIL ZINC OXIDE EUGENOL
	UNITS	ZONE OF INHIBITION IN MILLIMETERS*		
Penicillin sodium	2,000	20	20	20
Penicillin sodium	200	20	17	18
Penicillin sodium	20	17	13	14
Penicillin sodium	2	5	5	5
Zinc oxide and eugenol			6	

*Average of three tests.

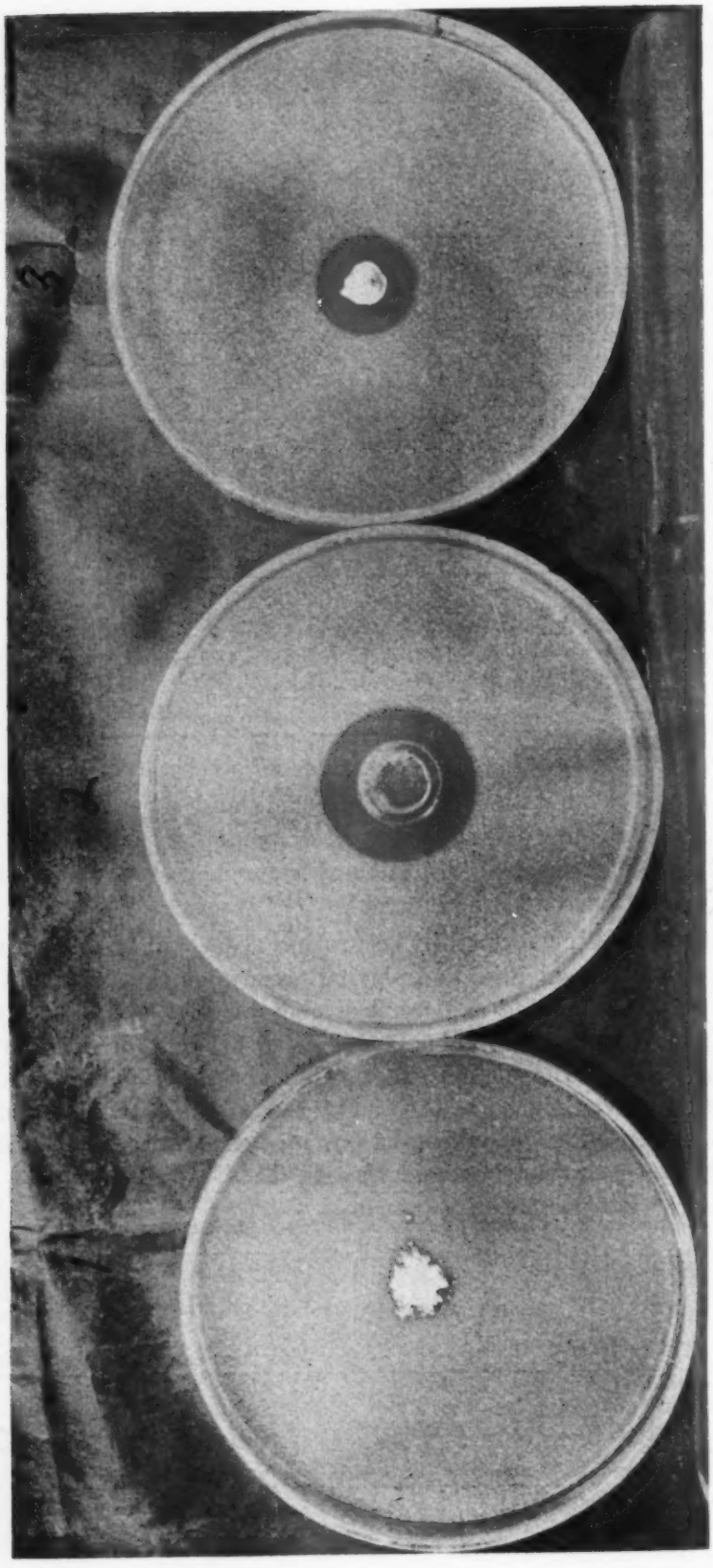


Fig. 1.—Infusion agar inoculated with *Staphylococcus aureus*. *1*, Zinc oxide powder, no restraint of growth; *2*, eugenol; and *3*, zinc oxide-eugenol, definite inhibition.

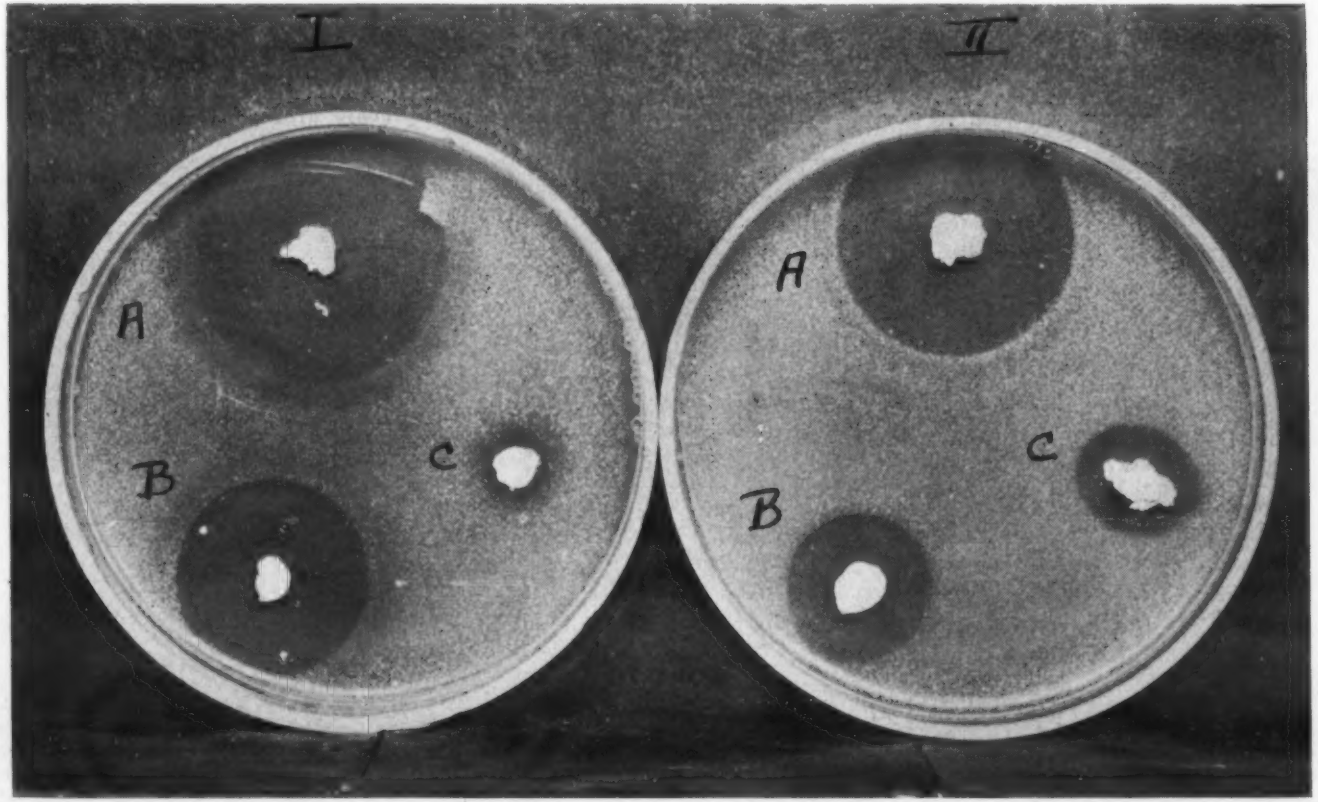


Fig. 2.—Infusion agar inoculated with *Staphylococcus aureus*. Effect of zinc oxide and zinc oxide-eugenol on concentrations of penicillin.

Plate I: *A*, Zinc oxide and penicillin, 200 units; *B*, with 20 units; *C*, with 2 units.

Plate II: *A*, Zinc oxide, eugenol, and penicillin, 200 units; *B*, with 20 units; *C*, with 2 units. (Inhibition in *C* probably due entirely to eugenol.)

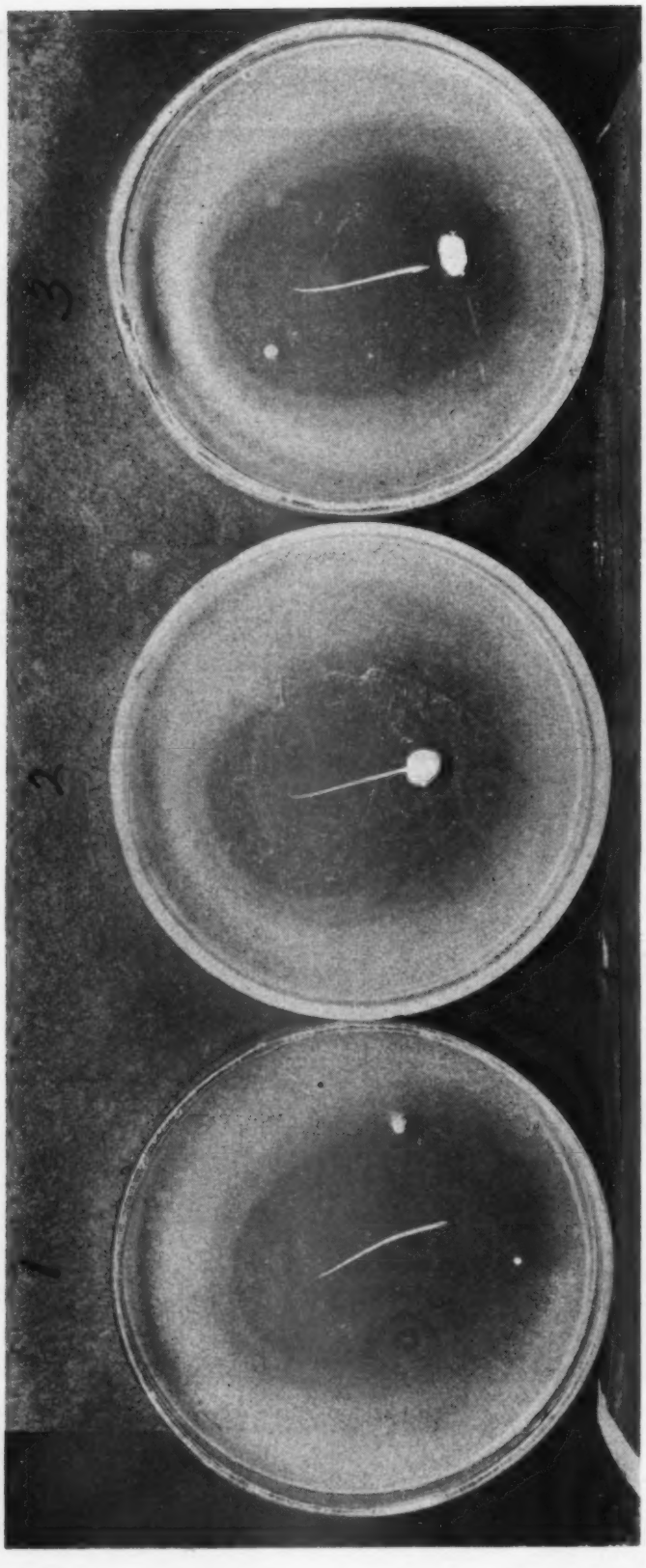


Fig. 3.—Infusion agar inoculated with *Staphylococcus aureus*. Effect of zinc oxide-eugenol on penicillin carried on absorbent points. *1*, Penicillin, 200 units; *2*, penicillin, 200 units, in contact with zinc oxide-eugenol; *3*, penicillin, 200 units, in contact mineral oil, zinc oxide-eugenol.

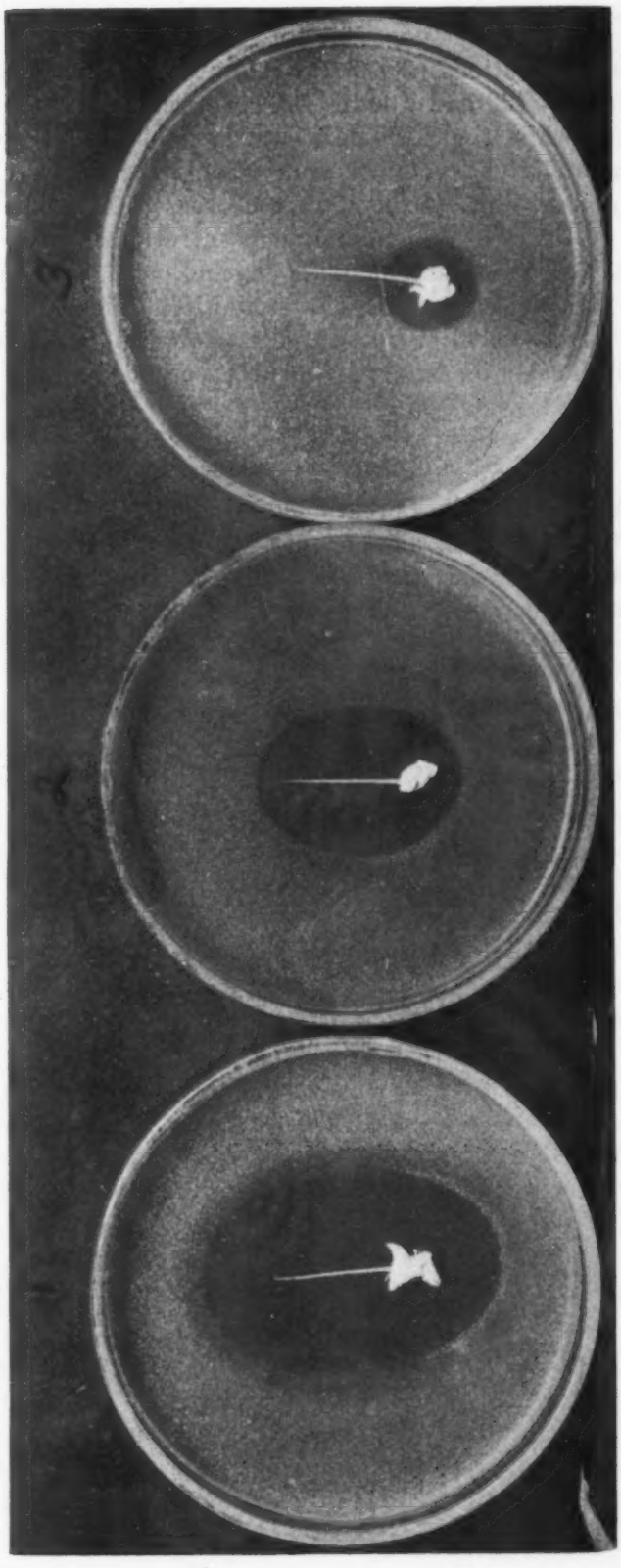


Fig. 4.—Infusion agar inoculated with *Staphylococcus aureus*. *1*, Penicillin 200 units, in contact zinc oxide-eugenol; *2*, 20 units, in contact zinc oxide-eugenol; *3*, 2 units in contact with zinc oxide-eugenol. (Inhibition of growth in *3* due probably entirely to eugenol.)

RESULTS

The higher concentrations (2,000 and 200 units) are not affected as much as the higher dilutions (20 and 2 units). This substantiates the results given in Table I. Thus, while the 2,000-unit saturated absorbent points show no decrease in their inhibitory potency even when exposed to zinc oxide and eugenol, the remaining dilutions did show a decrease. Yet sufficient penicillin was still active in the 200- and 20-unit concentrations to produce areas of bacteriostasis much larger than zinc oxide eugenol or eugenol alone produced. The 2-unit concentration causes areas of inhibition which in size are similar to those produced by eugenol or zinc oxide and eugenol, and therefore are at the limit of determination by this experimental method.

SUMMARY

Penicillin is affected by either eugenol or zinc oxide-eugenol cement. The extent in the reduction of the potency of low- and high-unit concentrations varies. Low-unit dilutions lose most or all of their inhibitory activity towards *Staphylococcus aureus,* whereas the higher-unit concentrations, though affected, still possess definite growth-restraining action.

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THE OROFACIAL CRIPPLE

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IT IS often within the province of the physician to make the initial suggestion to parents regarding the rehabilitation of their cleft palate child. Many parents are not cognizant of the fact that cleft palates can be repaired, either surgically or by a prosthesis. This seems to be due in some degree to an omission on the part of parents' health advisors. The attending physician has the opportunity to recommend appropriate treatment soon after the birth of a cleft palate child.

There are at least two reasons for instituting corrections of the palatal defect, as soon as technically possible, even though the repair may be of a temporary nature. First, the child's physical well-being will be better guarded and improved. The cleft, as a rule, makes feeding the child quite difficult, because there is interference with the intimate relationship of the anatomic structures forming and separating the nasopharynx and nasal cavity from the oropharynx and buccal cavity. As a result, the child is apt to be undernourished and its health may suffer. However, closing off the defect, even to a limited extent, will facilitate eating by the child, resulting in a better opportunity for normal development. Also, susceptibility to infection of the nasopharynx is possibly reduced.

Second, the morale and mental development of the growing child may be affected by the cleft. Experience indicates that the cleft palate child is an oral and facial cripple who often tends to react to mundane conditions in a manner similar to that of other types of cripples. Self-consciousness makes him hypersensitive to the taunts or the obvious pity of others. Such a child accepts his condition as a permanent inadequacy, or, on the other hand, his attitude may be one of open rebellion. In either case, he must struggle along under a handicap which usually interferes with his education and his social adjustments. He concentrates his attention on learning and play with difficulty. As part of his efforts, he employs compensatory, though abnormal, tongue and lip movements to disguise his conspicuous nasal tone, which results in incorrect speech habits. In any event, because of his handicap the patient often becomes a public charge. Therefore, early correction to obviate as much as possible of the physical and mental injury is of the utmost importance.

If the circumstances indicate that the defect should be corrected by mechanical rather than surgical procedures, the prosthetic correction can be obtained by means of an obturator. The latter is a disk, plate, or plug, natural or artificial, that closes an opening; or more specifically, from the dental standpoint, it is a denture that corrects an opening of the palate—soft, hard, or both.

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HISTORICAL REVIEW

One of the earliest appliances to be used in the mouth was the obturator. Although there is little doubt that persons afflicted with cleft palates improvised various simple and crude appliances such as wads of cloth, leather, and wax to lessen their handicap, the first definite record of the idea of an appliance so constructed as to close off a palatine defect was made by Alexander Petronius, in the fifteen century.[13]

In 1541, the first definite description of an obturator was published by Ambroise Paré.[14] His apparatus consisted essentially of a disk to fit over the defect, attached to a sponge placed through the defect into the nasal cavity. The sponge would swell when moistened by the surrounding secretions and thus hold the disk in place. Paré also constructed a cuff-button type of obturator with revolving stud. (Fig. 1.)

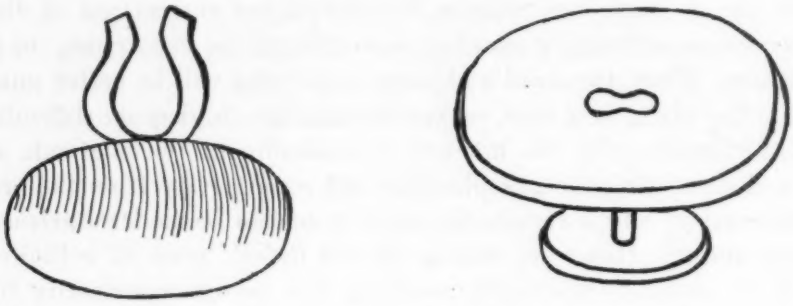


Fig. 1.—First illustrations of obturators by Paré in 1541.

The French were predominant in this field until the early nineteenth century. The French contributors included Pierre Fauchard, Bourdet, and Delabarre. Descriptions of the construction of obturators and discussions of their uses then began to appear in the English literature. The investigators in this field included Snell, Rowell, Hullihen, White, Suersen. The latter introduced the velum portion of the obturator. Kingsley did a vast amount of research work in this phase of dental prosthesis. From his researches he developed a fairly satisfactory technique for the correction of congenital cleft palate that was applicable to postoperative cases as well.

Among those contributing to the art of surgical prosthesis during the twentieth century are: Ackerman, who has done outstanding work in rehabilitating cancer patients; Bulbulian; Calvin Case; Eby and Kazanjian, who did much restorative work in World War I; Fitz-Gibbon, who has done much valuable work in speech rehabilitation of cleft palate patients; Lowery; Mitchell, who was the inventor of a specific type of hinged appliance that included the artificial vomer; Olinger; Ottolengui; and Young. Progress has been principally in the form of modification rather than originality, since the principles of design were established by the early workers. However, investigators have from time to time injected new designs and principles of technique, especially in speech-correction appliances. The rapid advances that dentistry has made in the

field of dental materials has made it possible to construct appliances that are lighter, more sanitary, and more suitable physiologically.

Obturators can be classified into two types, namely, those that correct acquired openings and those that correct congenital openings.

Obturators to Correct Acquired Openings.—Openings of the soft or hard palate may be the result of a number of conditions. Among them are trauma, for example a gunshot wound or the piercing of the palate with some sharp instrument such as a fragment of a steering wheel; the effects of various diseases, including syphilis, tuberculosis, osteomyelitis, and cancer; radium and x-ray burns, and surgical operation, wherein portions of varying size are removed from the palate. (Fig. 2.)

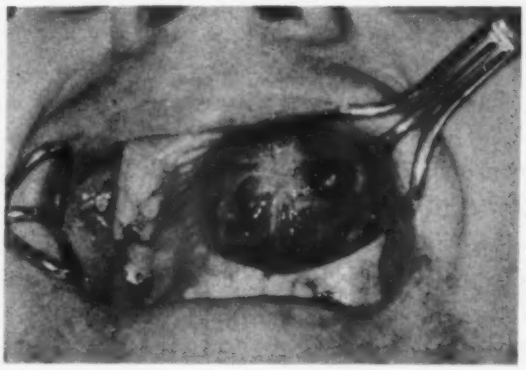


Fig. 2.

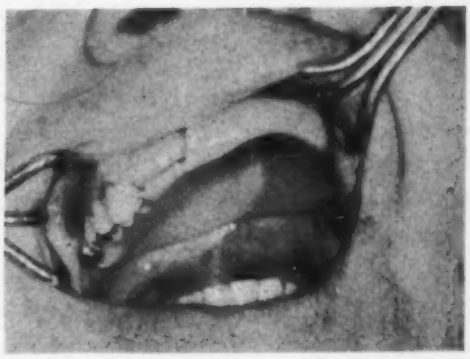


Fig. 3.

Fig. 2.—Palatal opening, result of surgical removal of neoplasm.
Fig. 3.—Temporary obturator for acquired defect.



Fig. 4.—Permanent obturator to correct an acquired palatal opening in an edentulous patient.

The defects produced by the various above-mentioned conditions may vary in size from less than a centimeter in diameter to an area that may include most of the hard palate, alveolar ridges, and floor of the nose. Therefore, the periphery of the defect may be of bone, soft tissue and bone, or only soft tissue. The lesion may occur anywhere on the palate.

The obturator may be constructed as a temporary or as a permanent appliance. With the temporary type the main objective is to close the opening. No endeavour is made to restore the patient's appearance. The temporary obturator usually does not include teeth in its construction. It may have stainless steel or gold clasps in dentulous cases. (Fig. 3.) While the apparatus is intended to serve only for a limited period, care should be exercised in its construction so as not to interfere with the regeneration of healing tissues. The temporary type is indicated where the patient is undergoing prolonged treatment, or where there is a possibility that the causing disease may recur. The obturator enables the patient to eat with greater comfort; it prevents food passing into the nasal cavity; it allows him to speak intelligibly, thus improving functional efficiency and morale.

The permanent type of obturator, as its name implies, is constructed to last indefinitely. It consists of durable materials, such as acrylic, chrome alloys, or gold. It includes teeth when indicated, to restore and improve functional occlusion and the appearance. (Fig. 4.) Speech is also improved. The construction of the permanent obturator is indicated when treatment has been concluded and the possibility of further surgical intervention in the wound area is remote.

Obturators to Correct Congenital Openings.—A cleft palate is a congenital fissure of the palate caused by arrested development of the associated developmental processes. Cleft palates may be divided into six types, the particular category depending on the portion of the palate found to be developmentally defective (Fig. 5). These are:

Class 1 Fissure of the azygos.
Class 2 Fissure in the uvula.
Class 3 Fissure in the soft palate.
Class 4 Fissure in the soft and hard palate.
Class 5 Fissure in the soft and hard palate through the alveolar ridge, at the junction of the maxilla and premaxilla, usually accompanied by a cleft lip on the same side.
Class 6 Fissure in the soft and hard palate through the alveolar ridge involving both side of the maxilla, freeing the premaxillary bone, and usually accompanied by a double cleft lip.

These categories can be further subdivided into the postoperative, the edentulous, and the postoperative edentulous.

Harelip may occur without a palate fissure, but the alveolar process is often involved.

The first two types are of little concern since they do not inconvenience the patient. Often the patient is unaware of these defects until they are disclosed during thorough mouth and throat examination. The remaining four types should have treatment, either surgical or prosthetic, or a combination of both.

The fixed obturator is one form of appliance utilized to correct congenital cleft palate, and consists of a palatal portion attached rigidly to a velar portion (Fig. 6). The principal types of fixed appliances are the solid obturator, the

Kingsley obturator, and the Fitz-Gibbon speech appliance. The first or solid type closes the defect by fitting into the nasopharynx and, by its walls, maintaining contact with the divided velum at all stages of phonation and deglutition. The Kingsley type has a flange contacting the divided velum, the portion attached to the plate being made of soft vulcanite rubber. The Fitz-Gibbon type is an all-metal appliance composed of a palatal plate, a tailpiece that corresponds to the normal soft palate at rest, and a bulb at the tailpiece whereby palatal function is simulated.

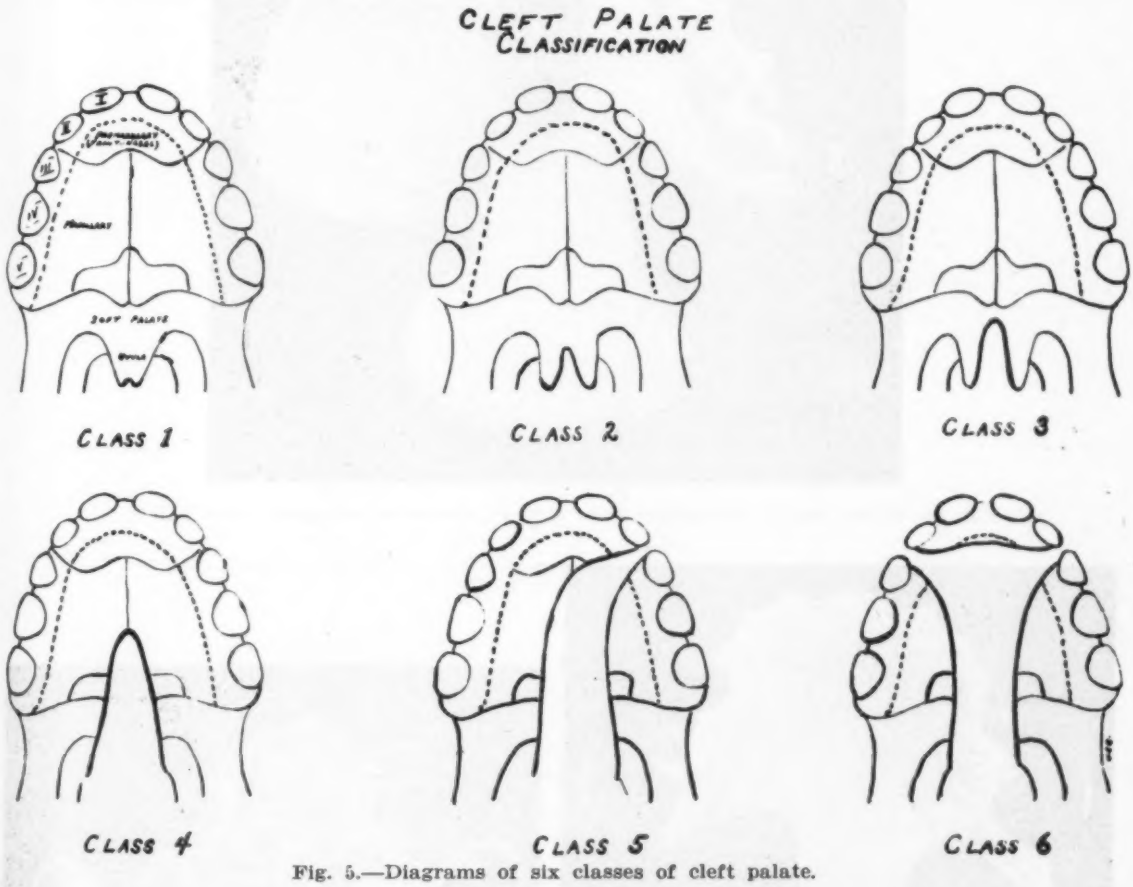


Fig. 5.—Diagrams of six classes of cleft palate.

The movable or pinlock hinge obturator is the other form of cleft correction appliance (Fig. 7). It is composed of the palatal portion, pinlock hinge, and artificial velum, and may or may not include a vomer portion. The movable type is indicated only where there remains an adequate flexible palatopharyngeal musculature.

Both the fixed and the movable type obturators are further subdivided into temporary and permanent types. The temporary appliance is constructed for children, whose jaws are changing and who have few, if any, erupted permanent teeth available for clasping (Fig. 8). In this case, the appliance consists of the palatal portion and clasps. The temporary appliance must be adjusted or renewed from time to time during the period the jaws are grow-



Fig. 6.—Fixed type of permanent obturator to correct a congenital cleft palate.

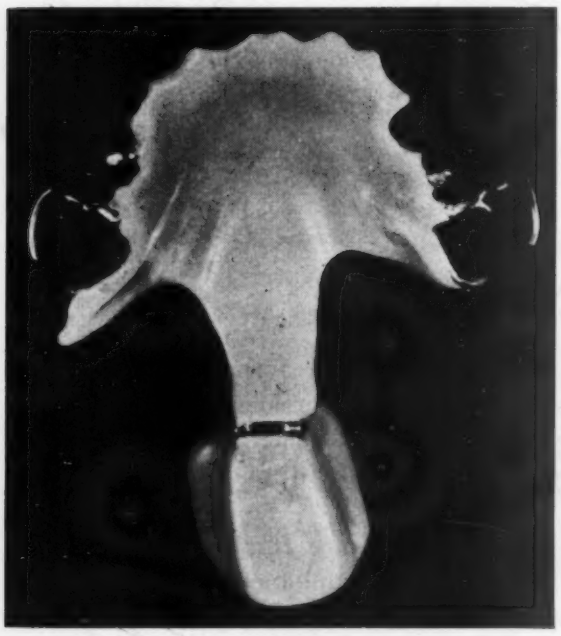


Fig. 7.

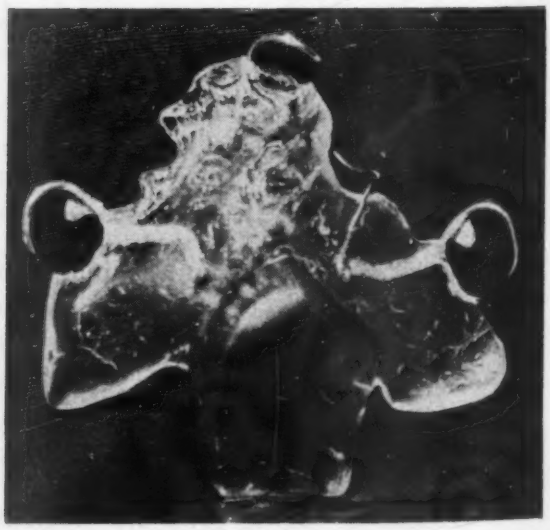


Fig. 8.

Fig. 7.—Movable or pinlock hinge obturator.

Fig. 8.—Temporary type obturator for cleft palate. Spring wire (*A*) is incorporated for tooth movement.

ing. After growth has reached an equilibrium, a permanent type may be constructed. If the pinlock hinge type is indicated, it consists of the above-mentioned movable parts that close off the two cavities in speech and deglutition, the velum being hugged and lifted by the remnants of the functioning palatal muscles until its posterior border contacts Passavant's pad* (Fig. 9).

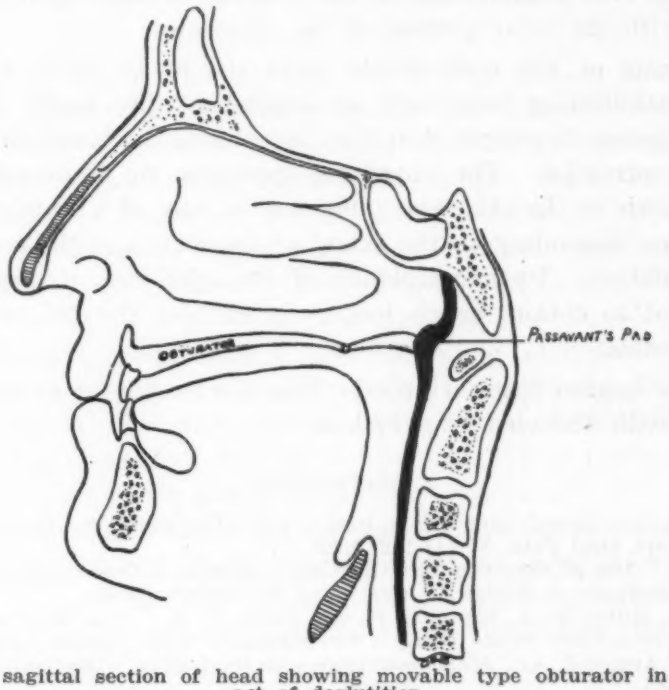


Fig. 9.—Median sagittal section of head showing movable type obturator in position in the act of deglutition.

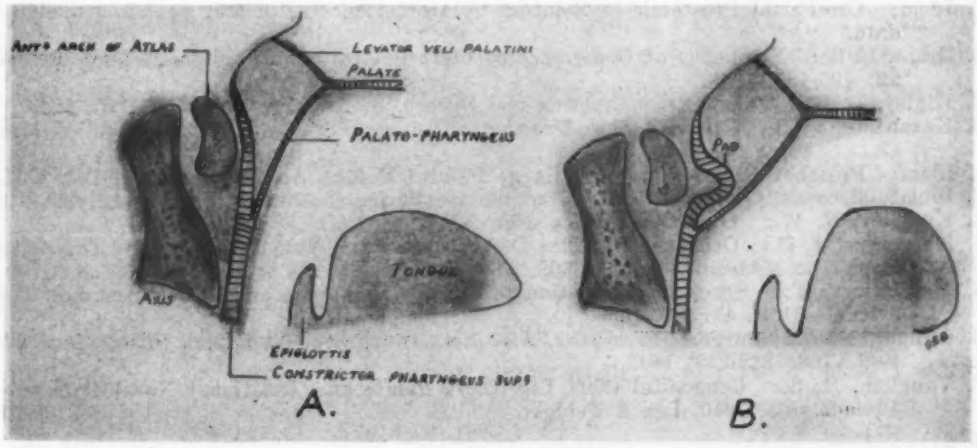


Fig. 10.—Formation of Passavant's pad (theory). *A*, Posterior sling relaxed. The superior constrictor muscle lies flat against the vertebrae, and the palatal fringes are at full length. *B*, The contraction of the palatopharyngeus muscle pulls up the constrictor muscle to form a cushion on the posterior pharyngeal wall.

*Passavant's pad is a cushion of superior constrictor muscle usually formed on the posterior pharyngeal wall at about the level of the tubercle of the first cervical vertebra, or atlas. One theory is that it is formed in the act of deglutition, etc., by the contraction of the palatopharyngeus muscle in pulling up the superior constrictor muscle to cause it to "bunch up" (Fig. 10).

Some patients will speak correctly in a very short time after the insertion of the obturator, but others will have incorrect speech habits which will make it advisable for them to attend a speech clinic. Here their problem will be analyzed by teachers experienced in the field, who will prescribe suitable corrective lessons and mouth exercises to strengthen the palatopharyngeal muscles. Usually this results in improved functioning of the muscles in their sphincter action in conjunction with the velar portion of the obturator.

The parents of the cleft palate child should be made to realize that means for rehabilitating their child are available. The family health advisor is the logical person to suggest that they investigate the possibilities of surgical or prosthetic correction. The prosthetic apparatus for improving speech and feeding is known as the obturator, and may be one of a number of different types of design, depending on the extent of the cleft and the condition of the palatal musculature. Upon completion of the appliance, it may be advisable for the patient to obtain speech lessons to exercise the palatal muscles and improve phonetics.

Let us not neglect these cripples! They are worthy of as much consideration as those with distorted arms or legs.

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POSTERIOR GROWTH OF MAXILLA

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PRESSURE or expansion forces are generally recognized in suture relationship of the skull bone. Exception to this view has previously been taken.

Pressure forces are also used to explain growth phenomena in nonsuture areas or closely approximating bones of the skull. Such an explanation is given to explain the posterior growth of the maxilla in the region of the tuberosities. The tuberosity region of the maxilla is presumed to articulate with the descending pterygoid process of the sphenoid bone during the entire period of its growth.

"The tuberosity rests against the descending pterygoid process of the sphenoid. This process grows downward and forward some 15 mm. before it stabilizes, so that the backward growth of the maxilla is actually transformed into a forward movement of the entire bone and this is further increased by the amount of growth of the pterygoid process.

"A little reflection reveals the necessity of the above arrangement. The backward growth, which amounts to the width of three molar teeth, would almost, if not completely close the pharynx, instead of permitting its enlargement. The pterygoid buttress keeps the rapidly expanding maxilla from encroaching on the pharynx, indeed shoves it forward."

The above conclusion is drawn from a study of the relationship existing between the tuberosities of the maxilla and the descending pterygoid processes of the sphenoid bone in the adult stage after growth has ceased. This relationship is presented in Fig. 1, where it is apparent that the posterior regions of the maxilla are directly anterior to the descending pterygoid processes and appear buttressed against them. This relationship, however, does not exist at any of the growing stages, as may be observed by a clinical examination of those regions of the skull at any stage of growth and development from birth to maturity.

Figs. 2, 3, 4, and 5 are lateral views of human skulls ranging from 6 months of age to 5½ years. The relationship between the growing posterior regions of the maxilla to the posterior regions of the skull are in each instance focused into view. It is apparent that during the growing stages the tuberosity regions of the maxilla are not directly anterior to the descending process of the sphenoid bone and are, therefore, not buttressed against them. The maxilla grows posteriorly into the sphenomaxillary space and the growing tuberosities are lateral and posterior to the descending pterygoid processes of the sphenoid bone. This finding is also presented in Figs. 6, 7, and 8, views of the base of the skull where both the lateral and posterior relationships may be observed.

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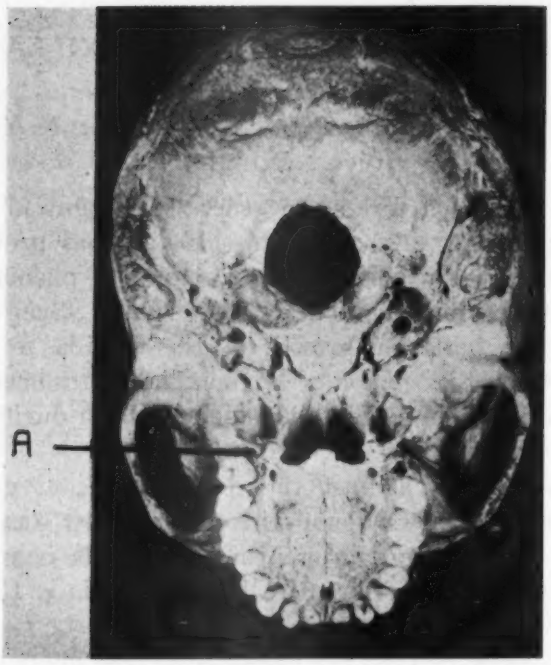


Fig. 1.

Figs. 1 to 9.—*A*, Posterior border of maxillary tuberosity. *B*, Descending pterygoid process of the sphenoid bone. *C*, Sphenomaxillary space.



Fig. 2.



Fig. 3.

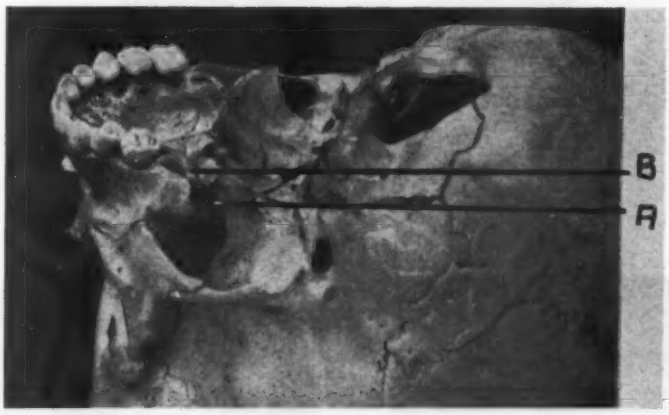


Fig. 4.



Fig. 5.

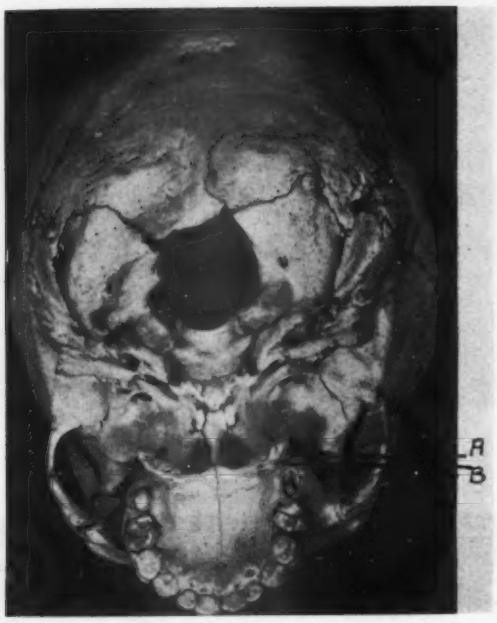


Fig. 6.

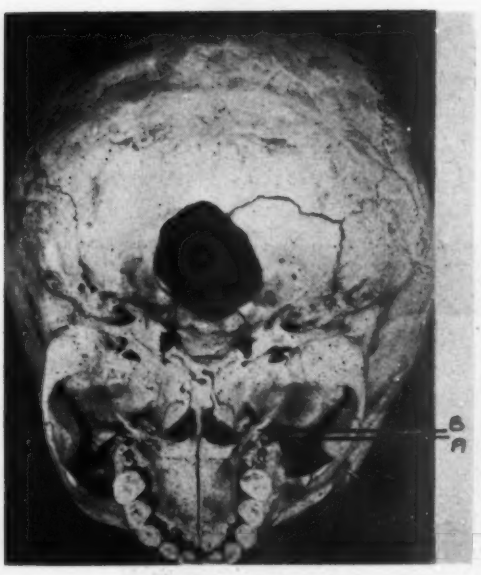


Fig. 7.

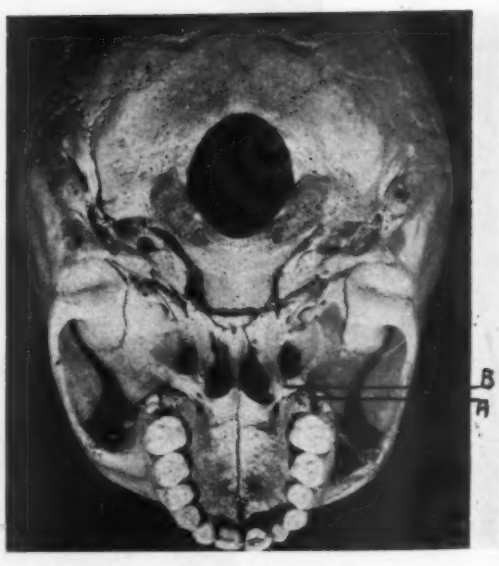


Fig. 8.

Considerable growth of the posterior regions of the maxilla has taken place between the stages presented in Fig. 6, of about 5 years of age, and Fig. 8, where the maxillary first permanent molar teeth are in clinical eruption, and yet the same anatomic relationships are maintained.

Fig. 9 is a histologic section of a human head about four months in utero. The same relationship may be observed in the embryonic and fetal stages as in the growing postnatal stages. At fetal stage there is no bone on the posterior border of the tuberosity of the maxilla as there is an absence of the posterior plate of bone during all the stages of the posterior growth of the maxilla. The posterior plate of bone is not formed until the last of the maxillary permanent molar teeth are formed, when growth in this area ceases.

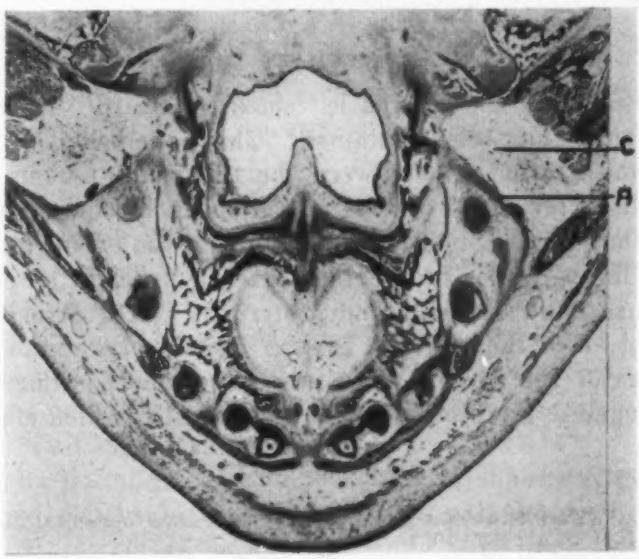


Fig. 9.

DISCUSSION

The explanation of pressure or expansion forces between adjacent bones in suture areas, or between adjacent approximating bone, has been resorted to because of a lack of proper consideration for the three-dimensional factor and the differential character of growth. The proper evaluation of both the three-dimensional factor and the differential character of growth reveals that sufficient spacial conditions are maintained to permit growth to occur without pressure or expansion forces. The synchronous interrelation of growth of adjacent structures is an important additional factor in this consideration. The accelerated stages of the posterior growth of the tuberosity region of the maxilla precede the accelerated intervals of growth of the descending pterygoid processes of the sphenoid bone.

Only after the growth of the posterior region of the maxilla is completed does the final accelerated stage of growth of the descending pterygoid processes occur in a lateral direction and assume a position more or less directly posterior to the tuberosity regions of the maxilla.

Another factor responsible for the belief that the maxilla is steadily "shoved forward" as growth occurs in the posterior region is the assumption of a stable or fixed point. It is necessarily assumed not only that the descending pterygoid plates are strong enough to resist the growing maxilla, but also that they remain, at least anteroposteriorly, in a fixed position. This obviously is not true. The base of the skull is constantly being remodeled and increasing considerably in size. Most of the additional area created is by remodeling of the occipital bone posteriorly. There is no danger, therefore, of the pharyngeal space becoming reduced in area because of the posterior growth of the maxilla.

One must keep other relations in mind as well. If the maxilla were "shoved forward," it necessarily would carry with it all of the bones articulated with the maxilla. These include all the bones of the face.

SUMMARY

The conclusion that the maxilla is "shoved forward" as growth occurs in the posterior region has been examined. This conclusion is based upon the anatomic relationships existing between the tuberosity regions of the maxilla and the descending pterygoid process of the sphenoid bone in the adult stage. It is assumed that this relationship exists during the entire growth history of these regions. Evidence has been presented that, instead, the posterior regions of the maxilla are found laterally and posteriorly to the descending pterygoid processes during all of the stages of growth from fetal stage to maturity. The posterior regions of the maxilla grow into the sphenomaxillary space and are not buttressed against the pterygoid processes until the final stage of maturity.

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THE CARIES PROBLEM

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LITERATURE

Tomes[1] was the first to describe the histologic alterations which take place in the carious tooth. He believed that the destructive changes in enamel were caused by the solvent action of acid from the oral fluids. He also described changes in the dentin, calling attention to the transparent zone.

The pioneering work of Leber and Rottenstein[2] laid the foundation for the chemicoparasitic theory of W. D. Miller.[3] Miller described the initial attack on enamel as a dissolution of the interprismatic substance by acid, produced by microorganisms on the enamel surface. His experiment consisted in placing pieces of teeth in a mixture of saliva and bread. "In many pieces the dentin was softened through and through. When the softening had penetrated through the dentin to the inner surface of the enamel, the latter was found covered with a layer of white powder exactly as it is found in natural decay." He further states, "Where the enamel was hard and thick without cracks or other defects it had not even lost its natural polish . . . These experiments show among other things, to what an extent the resistance which a tooth opposes to the destroying factors depends upon the structure."

Williams[4] later described the bacterial film or plaque attached to the enamel in incipient caries. Miller also showed microorganisms present in carious dentin. He stated that dental caries consists of two stages: first, decalcification or softening of the tissue, and then proteolysis of the softened residue.

For the past twenty years, most of the interest in the bacteriology of dental caries has centered in the lactobacilli.[5] Even though lactobacilli may play an important part in the etiology of dental caries, all the evidence available today indicates that no one species or strain is responsible. The evidence also favors the view that dental caries is not a specific bacterial disease, as was postulated by Miller,[3] Goadby,[6] and Kantorowicz.[7] The work of McIntosh, et al.,[8] suggests that perhaps one and the same organism is either acidogenic or proteolytic, as conditions change during bacterial penetration of enamel and dentin. The paradox of undermining caries may be explained by accelerated bacterial activity under anaerobic conditions in the dentin.

Several investigators described microorganisms in decalcified sections of enamel shortly after Bodecker[9] offered a special method for preservation of the organic matrix. Baumgartner[10] and Fleischman[11] claimed that, after penetrating the enamel sheaths, bacteria produce acid in situ and cause decalcification. Malleson[12] showed bacteria working their way between prisms in the enamel.

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Bodecker[13] found bacteria in the enamel rods following acid dissolution of the inorganic salts.

Gottlieb[14] first suggested that the carious process in the enamel begins as a proteolysis of organic structures, especially the unhornified cuticle and lamellae. He also claimed that acid is secondarily involved in the process. He later found[15] a yellow pigmentation characterizing the proteolytic process in all its stages. Heider and Wedl[25] in 1864 described and illustrated in color the yellowish pigmentation associated with caries. Gottlieb, however, made his observations independently. Pincus[16] and Frisbie, et al.,[17] have also supported the concept of proteolytic action in caries.

Gottlieb recommended a special silver nitrate preparation as a prophylactic against caries, on the ground that it impregnates or blocks the organic roads of bacterial invasion. Previous investigators for nearly a century had also recommended it, but on the assumption that it inhibits the action of acid on the inorganic elements of enamel.

Beust[18] observed that if you take a tooth with incipient caries and immerse it in lactic acid, the sound enamel will be decalcified and the decayed part will remain. Beust concluded that something else is necessary for caries progress besides acid—possibly the direct action of enzymes. Malleson[12] wrote, "It might be anticipated that enamel already partially decalcified by bacterial action and then subjected to the action of acids in process of preparation for section cutting would be still more difficult to stain. This is not so and it would seem as if the organic content of affected enamel were in some way increased. How this is brought about is difficult to determine. It may be a direct effect of bacterial infection or it may be that the proteids of the saliva penetrate the enamel prisms as these are partially decalcified."

PROCEDURE

Teeth were selected representing various types of carious lesions, from teeth apparently intact, except for evidence of small areas of brown discoloration on the enamel surfaces, to teeth showing large carious lesions with cavity formation. These were prepared for study, partly in ground sections and partly in decalcified sections. In most instances photomicrographs were taken, both in black and white and in color. The ground sections were also studied by means of the grenz ray.[19] The authors recognized a special value in a comparative study of the histologic and grenz-ray methods. The grenz ray discloses variations in calcification of the hard tissues more sensitively than the x-ray, which is commonly used clinically to detect carious lesions. The histologic procedures reveal phenomena which cannot be observed by the grenz-ray method. In selecting the material for study we tried to find a series of specimens which showed various stages of the carious process. In this way, we believed it possible to trace the pathogenesis of caries sequentially, to the end product or the cavity.

FINDINGS

Fig. I is a study of a ground section, with a carious lesion in the intercuspal region of a maxillary permanent molar. Clinically the lesion appeared as a characteristic brown spot with no break in the enamel.

A. The black and white photomicrograph of the lesion. It shows a definite variation in pigmentation. The regions closer to the occlusal surface of the enamel are quite dark; the deeper area is light gray. The lesion does not extend to the dentino-enamel junction, and there is no evidence of changes in the dentin.

B. The color photomicrograph of the same lesion. The occlusal half is dark brown and the remainder of the lesion light brown or amber. The lesion does not extend to dentino-enamel junction and there is no evidence of dentin involvement.

C. A grenz-ray negative of the same lesion. It shows the distribution and variation in degree of radiolucent areas in the enamel.

Fig. II is a ground section of an area of enamel which clinically showed a characteristic brown spot with no break in the enamel.

A. The black and white photomicrograph of the lesion and surrounding areas. The region near the peripheral surface appears quite dark in the center of a cone-shaped light-gray area. A dark streak traverses the lesion in a linear direction, and terminates at the base of the lesion. There is no evidence of change in the dentin.

B. The colored photomicrograph of the same lesion. The area of the periphery of the enamel is dark brown, surrounded by a cone-shaped light-brown pigmentation. A dark line extends in a linear direction to the base of the lesion, which is some distance from the dentino-enamel junction. The dentin appears intact.

C. A grenz-ray negative of the same lesion. It shows a radiolucent area of varying density in the peripheral half of the enamel, with a peripheral border of radiopacity. A dark streak extends linearly to the base of the lesion.

Fig. III is a ground section of an area of enamel and dentin of a tooth which showed a characteristic brown spot, with no evidence of a break in the enamel.

A. The black and white photomicrograph of the specimen. The lesion appears fairly black in the peripheral half of the enamel, with a light area at the periphery of the lesion. At the base of the lesion there is a fringe of variegated gray discoloration. A dark linear band runs from the base of the lesion, which does not quite communicate with the enamel tuft. A number of tufts are in evidence at the dentino-enamel junction. The underlying dentin is normal.

B. A colored photomicrograph of the lesion and the surrounding enamel and dentin areas. The lesion in general appears largely as light brown, interspersed with small dark-brown areas. Around the base of the lesion, extending about two-thirds of its periphery, is a band of dark brown, almost black, beyond which is a fringe of very light brown. An area near the periphery of the lesion is light brown. A linear, dark band extends from the base of the lesion and almost communicates with an enamel tuft. A number of tufts are in evidence near the dentino-enamel junction. The underlying dentin appears normal.

C. The grenz-ray negative of the same area, showing a radiolucent area in the peripheral half of the enamel, with a radiopaque area at the periphery and a fringe of slight radiolucency at the base of the lesion.

Fig. IV is a ground section of a tooth showing an area of enamel and dentin. Clinically this area presented a brown spot, without any break in the enamel.

A. The black and white photomicrograph of the area, disclosing a wedge-shaped lesion in the enamel extending from the periphery almost directly to the dentino-enamel junction. The peripheral margins of this area appear quite dark, and the center fairly light, except for isolated dark regions. A fringelike border of lighter shade surrounds the deepest part of the lesion. Three dark streaks radiate from one side of the lesion to the dentino-enamel junction. Directly under the base of the lesion the dentino-enamel junction appears dark, as does a semioval zone of underlying dentin. On the one side, the dentino-enamel junction appears wide, and is formed by a series of scalloped layers. A number of tufts radiate into the enamel.

B. A colored photomicrograph of the same area. The lesion in the enamel appears in its periphery as a dark brown band. The center of the lesion is light brown or amber except for one area, which is dark brown like the periphery. The lesion at its base extends very close to the dentino-enamel junction, and the fringelike border reaches to it. Three brownish streaks radiate from one side of the lesion directly to the dentino-enamel junction. The dentin directly under the base of the lesion is a yellowish-orange; this is surrounded by a light zone.

C. A grenz-ray negative of the same area. The area of the lesion appears slightly radiolucent, except for a small area at the side, which is more radiolucent. The enamel at the base of the lesion appears radiopaque. A semioval area of dentin directly underneath the lesion appears radiolucent.

Fig. V is a ground section of a molar tooth showing a small area of enamel between two abraded cusps, denuded of enamel.

A. The black and white photomicrograph of the enamel and adjacent areas. A dark wide band is seen running from the summit of the enamel through its central area to the dentino-enamel junction. Adjacent to the band on either side are localized areas which appear less dark than the central band. A fairly extensive area of dentine directly underneath the enamel appears generally light, but with some dark or translucent areas.

B. A kodachrome photomicrograph of the same specimen. A dark brown, almost black, band extends through the center of the enamel from the periphery to the dentino-enamel junction. On either side of this band are areas in varying shades of yellowish-orange. The area of dentin directly underneath the enamel presents an area of yellowish-orange pigmentation in its center, surrounded by areas of dark brown followed by a light band.

C. A grenz-ray negative of the same specimen. The enamel appears radiopaque, with a dark or radiolucent band running from the periphery through the center, but not reaching the dentino-enamel junction. Directly under the enamel a large area of dentin appears radiolucent.

Fig. VI is a ground section of an area of enamel and dentin. The clinical examination disclosed a dark brown area, with no break in the enamel.

A. The black and white photomicrograph of the specimen. In the central area of the enamel there is a black rectangular mass which extends from the periphery to the dentino-enamel junction. The dentino-enamel junction adjacent to this black area is irregular. An extensive area of dentin directly underneath the enamel appears generally light, with a dark band surrounding a central area a short distance from the periphery.

B. A kodachrome photomicrograph of the same area. The central area of the enamel appears dark brown to black. The area of the dentino-enamel junction and dentin directly adjacent is yellowish-orange, delimited by a brown band. An area of dentin directly underneath this brown band is light brown in color; this area is surrounded by a band of darker brown. Adjacent to this is a translucent zone.

C. A grenz-ray negative of the same area. The area of the lesion is bordered on each side by a radiolucent line. Except for a small radiolucent depression at the periphery, the area of the lesion is slightly radiolucent, hardly to be differentiated from the normal radiopaque enamel on either side of the lesion. The dentin directly underneath the enamel is markedly radiolucent, with a larger area of lesser radiolucency extending deeper in the dentin. This is surrounded by a radiopaque band.

Fig. VII is a ground section of a tooth showing an area of enamel and dentin. A characteristic brown spot was tested clinically with no break in the enamel.

A. The black and white photomicrograph of the area, showing a large, half-round black area, which extends from the periphery of the enamel to about one-third its depth in the direction of the dentino-enamel junction. A dark band extends from one side of this area as far as the dentino-enamel junction and slightly beyond the dentin. A series

of tufts extend from the dentino-enamel junction into the enamel. A fairly deep, half-round area of the underlying dentin appears translucent.

B. A kodachrome photomicrograph of the same area. The enamel generally appears light brown. A half-round area extending from the periphery of the enamel appears black in its peripheral half, and orange-brown in its deeper half. A brown band extends through the orange-brown area and beyond as far as the dentino-enamel junction and a slight distance into the dentin. Tufts extend from the dentino-enamel border a short distance into the enamel. An area of the enamel adjacent to the area previously described appears brown, and extends almost to the dentino-enamel junction. The area of dentin adjacent to the dentino-enamel junction appears translucent.

C. A grenz-ray negative of the same lesion. The enamel appears generally radiopaque except for a thin radiolucent line which extends from the periphery of the enamel to the dentino-enamel junction. Surrounding this radiolucent line in the region toward the periphery of the enamel, the area of enamel is very slightly radiolucent. The dentin immediately adjacent to the dentino-enamel junction appears radiolucent.

Fig. VIII is a ground section of a tooth showing an area of enamel and dentin. A characteristic brown spot was seen clinically with no break in the enamel.

A. The black and white photomicrograph of the area of dentin and enamel, showing a dark band extending from the periphery of the enamel to the dentino-enamel junction and slightly beyond into the dentin. On either side of this dark band, in the peripheral half of the enamel, appears an area stippled with small gray spots. There are tufts all along the dentino-enamel junction. The area of dentin adjacent to the dentino-enamel junction appears translucent.

B. A kodachrome photomicrograph of the same area. A dark band is seen extending from the periphery to the dentino-enamel junction and slightly beyond. The enamel appears generally light brown, with the exception of the peripheral third of the enamel. An area surrounding the dark band in the peripheral third of the enamel may be differentiated from the rest of the enamel. Enamel tufts are present along the dentino-enamel junction. Immediately surrounding the dentin terminal end of the dark line, is a yellowish-orange area in the dentin. This is surrounded by a light or translucent area.

C. A grenz-ray negative of the same area. The enamel appears generally radiopaque, with the exception of a radiolucent line extending from the periphery to the dentino-enamel junction. The area of enamel immediately surrounding the thin radiolucent line in the peripheral third is slightly radiolucent. The dentin directly adjacent to the dentino-enamel junction is radiolucent.

Fig. IX is a ground section of a tooth showing an area of enamel and dentin. Clinically there was no break in the enamel.

A. A black and white photomicrograph of this area is not published.

B. A kodachrome photomicrograph of the area. The enamel appears regularly light brown in color, with two dark-brown, almost black, streaks running from the periphery to the dentino-enamel junction and slightly beyond into the dentin. The dark streak to the right is an artifact. A large yellowish-orange area is seen in the dentin, extending from the dentino-enamel junction and including the terminal ends of the two dark bands. Surrounding this yellowish-orange area the dentin appears brown.

C. A grenz-ray negative of the same area. The enamel appears generally radiopaque, with the exception of two radiolucent lines, one of which extends from the dentino-enamel junction to the periphery, and the other from the dentino-enamel junction about two-thirds the width of the enamel. A round radiolucent area extends from the dentino-enamel junction into the dentin, with a radiolucent extension radiating from it deeper into the dentin.

Fig. X is a ground section of a carious tooth showing a cavity surrounded by enamel and dentin.

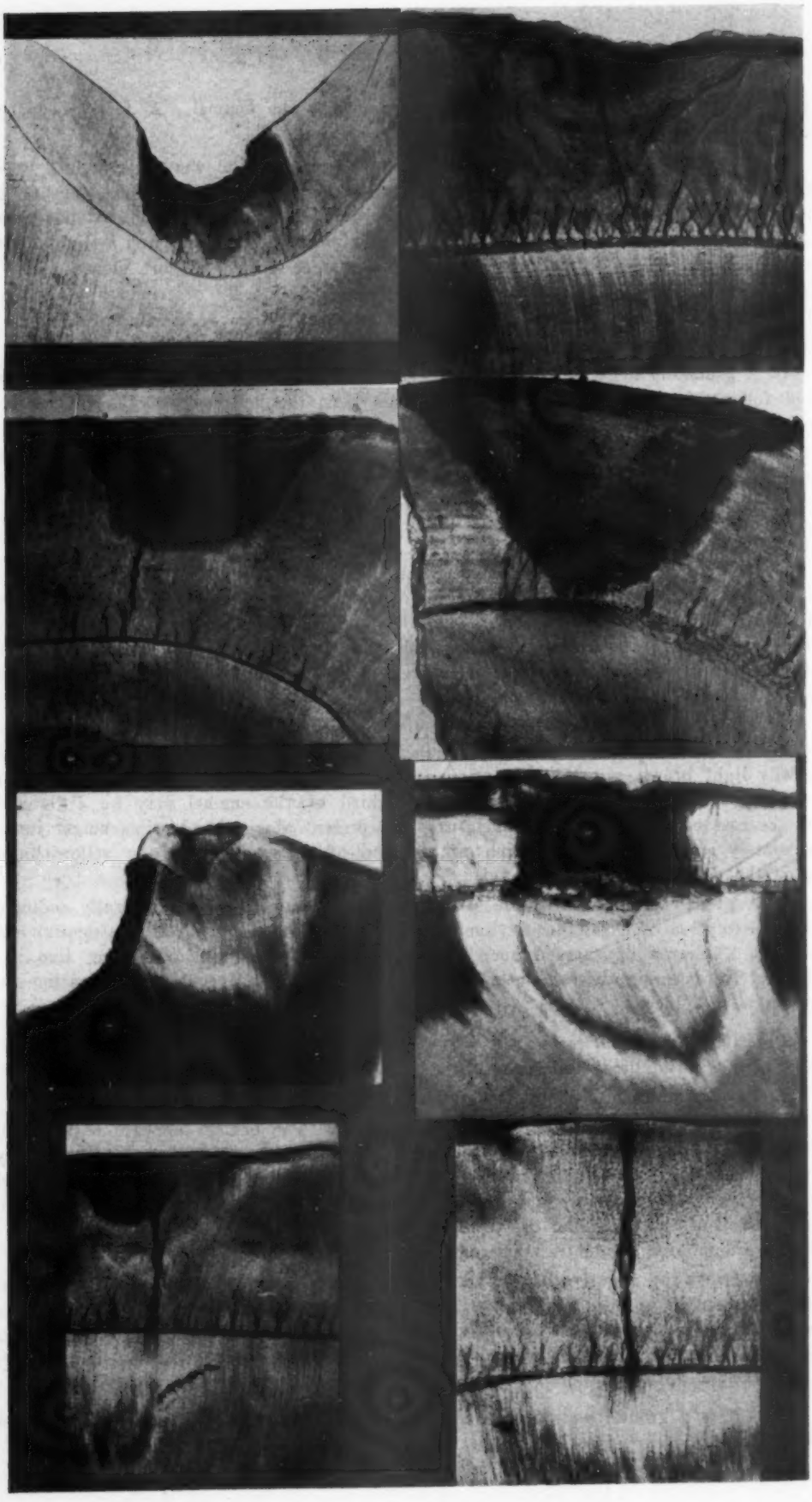
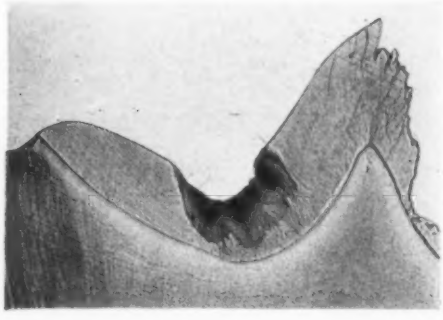
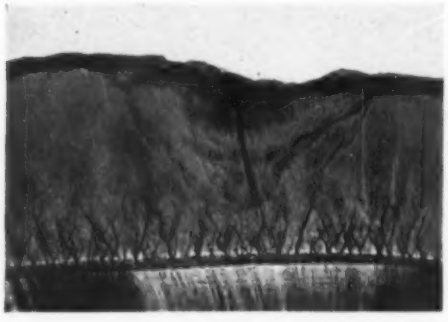


Plate A.

I



II



III



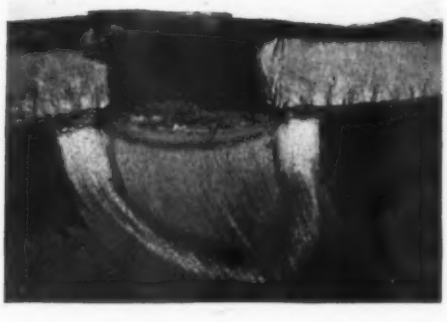
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V



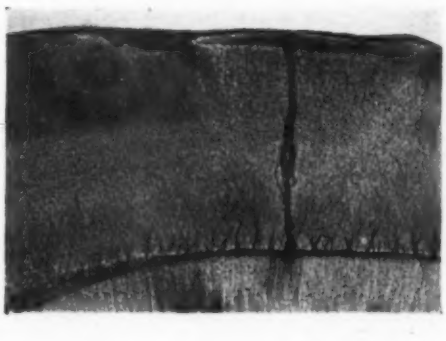
VI



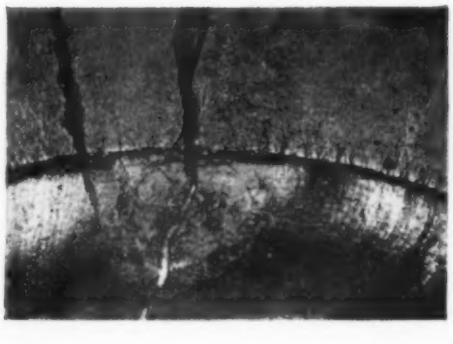
VII



VIII



IX



X



PLATE B.

(Kodachromes by Julius Weber, Columbia University.)

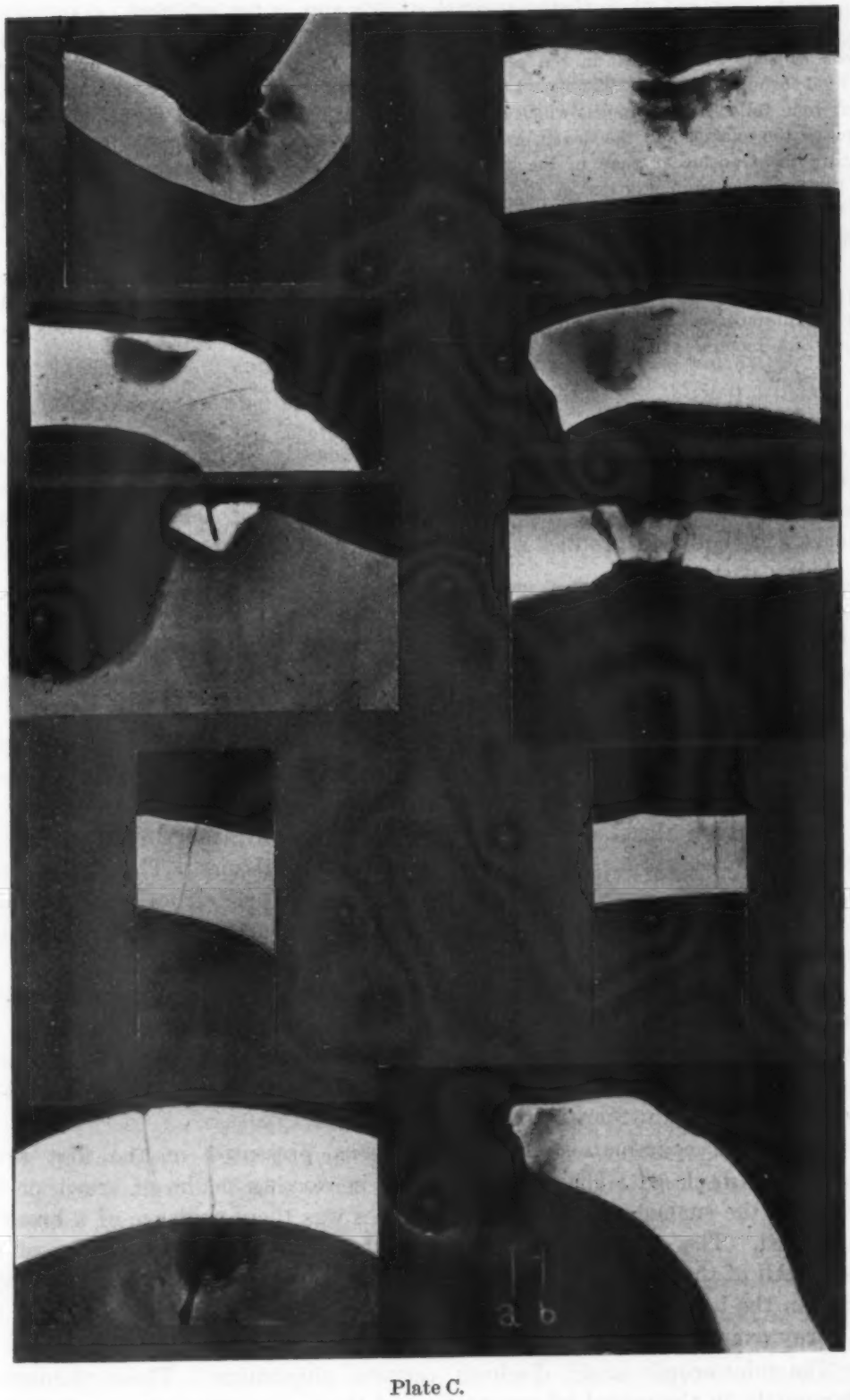


Plate C.

A. A black and white photomicrograph of this area is not published.

B. A kodachrome photomicrograph of the area. The enamel adjacent to the carious cavity appears dark brown in the peripheral third. The dentinal two-thirds of the enamel is light brown in color. This whole area is delimited from the rest of the enamel by a light zone followed by enamel, which appears regularly light brown. A slight space is noted between the enamel and the dentin extending from the carious cavity to the region approximating the translucent zone in the enamel. Directly underneath the clear space is a zone of light orange pigmentation in the dentin. The deeper dentin appears in various shades of brown. Laterally there is seen another elliptical zone of orange brown with a light border followed by a dark brown border.

C. A grenz-ray negative of the same area. The enamel appears radiopaque, except for a small area of the enamel adjacent to the carious cavity. This area appears radiolucent, interspersed with radiopaque streaks. The dentin underlying the carious cavity appears radiopaque at A, followed by a radiolucent zone at B.

DISCUSSION

Acid and Proteolytic Action.—It can readily be demonstrated experimentally that acid alone, in whatever concentration, acting upon mature enamel, does not produce true caries. Slight concentration of acid, used over short intervals of time, will produce a characteristic phenomenon frequently described as chalky enamel.

It is our belief that chalky enamel lesions, as seen clinically, are not evidence of true caries, but merely evidence of dissolution of calcium salts. They may be associated with the caries phenomenon, either preceding it or accompanying it. Whatever part acid action may play in the caries process, it is apparent that another factor must be present to produce the disease of caries. This factor is concerned with the destruction of the organic substance, resulting in necrosis, its final stage. This factor must necessarily be proteolytic in nature.

Acid action alone, especially in the low concentration tolerated physiologically, cannot produce necrosis of organic hard tissues. The organic content would resist complete destruction by even much stronger acids, as is commonly demonstrated in the decalcification technique for histologic investigation of hard tissues.

In a previous study by one of us,[15] evidence was presented that the earliest sign of the true carious process is the appearance of a yellow pigmentation. At such an early stage, the histologic structure of the affected tissue is still recognizable. It was also observed that the yellow pigmentation was more evident in the dentin than in the enamel.

A clinical examination of the specimens presented in the first seven illustrations disclosed a characteristic lesion in varying shades of brown on the surface of the enamel. In none of these cases was there evidence of a break in the enamel. The ground sections were made through the central area of the lesion. All of these lesions would generally be diagnosed as evidence of arrested caries on the basis of clinical examination; and, with the exception of Fig. VI, the x-ray examination would be negative.

The microscopic study discloses varying phenomena. These phenomena differ partly in the extent of penetration of the lesion into the enamel, as seen

in the black and white photomicrographs, and partly in a striking variation of pigmentation in different regions of the same lesion, as observed in the color photomicrographs. These studies also reveal that some of these enamel lesions are associated with the presence of lamellae, while others are not.

Figs. I, II, and III present lesions of the enamel which do not extend to the dentino-enamel junction. The black and white photomicrographs of Figs. I and II disclose that the peripheral areas of the lesion are quite dark, whereas the deeper parts of the lesion and the lateral borders are fairly light gray. In each instance a lamella appears to extend through the lesion but does not reach as far as the dentino-enamel junction. In Fig. II, while the enamel adjacent to the dentin is profuse with tufts, the lesion does not appear to extend as far as the terminal ends of the tufts. The dentin appears normal.

The kodachrome photomicrographs compare favorably with the black and whites. The areas of the lesion which appear dark, or black in the black and white photomicrographs, are dark brown in the kodachrome, and the areas which appear light gray are light amber. The grenz-ray study of these lesions discloses evidence of decalcification of the enamel to a degree varying with the pigmentation. Those areas of the lesion which appear dark or black in the black and white photomicrograph or dark brown in the color photomicrograph show evidence of considerable decalcification in the grenz-ray negative, whereas the lighter areas reveal comparatively slight decalcification. The areas of slight changes in pigmentation as seen in the black and white and color photomicrographs show no decalcification of the enamel in the grenz-ray study. The distribution of decalcification areas as seen in the grenz-ray negative is therefore comparable in degree and extent of decalcification to the degree of increased pigmentation of the enamel lesion.

Similar findings are noted in Fig. III, where the entire lesion, except for a small area near the periphery, is quite dark or black in the black and white photomicrograph, and dark brown in the color photomicrograph. The grenz ray reveals an area of decalcification comparable to the dark pigmented regions, with no evidence of decalcification in the peripheral region of the lesion, which appeared light gray in the black and white photomicrograph and amber in the kodachrome. It is interesting to observe, too, a light gray, fringelike border at the deepest part of the lesion. This may also be observed in the kodachrome as yellowish-orange. It is apparent from the study of the lesions in these specimens that the decalcification process of the enamel associated with caries is a very slow process and indicates an exceedingly low acid concentration. The light areas indicate a continuing penetration of the caries lesion deeper in the enamel.

The very light terminal borders of the lesion must therefore indicate the most recent stages of penetration of the pathogenic process deeper into the enamel. At this incipient stage, it is apparent that, if acid is present, it is not in sufficient concentration to give evidence of decalcification. The dark brown regions of the lesion, which give evidence, in the grenz-ray study, of decalcification, indicate necessarily a greater acid concentration, although not sufficient to produce complete dissolution of the enamel.

It is important to try to explain the dark brown pigmentation. It has been demonstrated that the enamel matrix contains as much as 33⅓ per cent of the total amount of inorganic salts found in mature enamel. A 5 per cent concentration of acid will dissolve the inorganic salts present and transform the enamel matrix from an opaque white to a dark brown. It is therefore conceivable that acid associated with enamel caries will in time produce similar effects. It is conceivable that the deeper, lightly pigmented, borders are indications of a continuing active process, and that these light areas would in time become dark brown and would give evidence of greater decalcification. The short, fringelike, but dark border of the lesion in Fig. III would indicate a much slower rate of deeper penetration than is indicated by the wide and lightly pigmented borders of the lesions presented in Figs. I and II.

An additional observation associated with the enamel lesions presented in Figs. I, II, and III is the absence of pathogenic lesions in the underlying dentin. This was observed not only in these three specimens presented, but also in a larger amount of material studied, in which the lesion extended to within some distance of the dentino-enamel junction.

As soon as the lesion reaches the dentino-enamel junction, however, as seen in Figs. IV, V, and VI, a characteristic change, recognizable as caries of the underlying dentin, is observed. This change is characterized by the yellowish-orange pigmentation, usually surrounded by a translucent or light zone. In Fig. IV-B the yellowish-orange pigmentation not only underlies the enamel lesion, but extends as far as the terminal ends of the three lateral lamellae. In Fig. IV-C the grenz-ray negative shows the characteristic distribution of areas of decalcification of the enamel in relation to the degree of brown pigmentation of the enamel lesion.

In the first three specimens (Figs. I, II, and III), a fairly definite correlation was established between the degree of pigmentation and the degree of decalcification as seen in their respective grenz-ray negatives. This correlation is not as definite in the lesion presented in Fig. IV, as may be seen by a comparison of the histologic and the grenz-ray study. The dark, fringelike border of the lesion, as seen in Fig. IV-A and IV-B, gives no evidence of decalcification in the grenz-ray negative. Accordingly, there is a zone of enamel, underneath the decalcified area, which appears to be as radiopaque as the adjacent normal enamel. This lack of correlation is even more striking in Fig. V.

Fig. V-A and V-B reveal a fairly wide, dark-stained fissure, extending from the periphery of the small piece of enamel to the dentino-enamel junction. On either side of the fissure, the enamel fragment reveals darkly pigmented zones. Yet the grenz-ray negative shows no evidence of decalcification of the enamel, except for the fissure area, which appears radiolucent. The fissure does not quite extend to the dentino-enamel junction. It is apparent that the dentin caries of the specimen in Fig. V had its direct source of infection through the enamel, and not from the lateral dentin regions of the abraded cusps. These not only appear to be intact but also give evidence of the characteristic aging phenomenon of dentin associated with mechanical stresses and impregnation of oral secretion.

A still more striking lack of correlation between a dark pigmented lesion and its grenz-ray negative is observed in Fig. VI. In the specimen Fig. VI-A, the black and white photomicrograph presents an enamel lesion quite dark in appearance and extending from the surface at a wide base directly to the dentin junction. The dark lines adjacent to the lesion are artifacts or cracks produced during the grinding of the section; and it is evident that the area of the enamel involved with the lesion is slightly misplaced peripherally from the rest of the enamel. The kodachrome, Fig. VI-B, reveals the enamel lesion to be dark brown. But the grenz-ray negative (Fig. VI-C) unexpectedly reveals that, with the exception of one small isolated area, there is only slight evidence of decalcification of the enamel.

The kodachrome (Fig. VI-B) shows a more advanced state of dentin involvement, with the characteristic yellowish-orange pigmentation, surrounded by a translucent or light zone. The evidence as determined by the grenz-ray criterion (Figs. IV, V, and VI) of no decalcification or negligible decalcification of lesions, which by histologic examination are revealed as enamel caries, leads to the conclusion that acid formation is not a necessary precursor of enamel caries formation.

The evidence so far presented indicates that, while in some caries lesions enamel decalcification occurs as a component of the caries process, in others there is only negligible decalcification.

This conclusion is considerably strengthened by additional evidence presented in Figs. VII, VIII, and IX. Figs. VII-A and VII-B, presents a characteristic enamel lesion extending only about one-fourth the depth of the enamel from its periphery. The rest of the enamel, as far as the dentino-enamel junction, appears normal. A lamella extends from the base of the enamel lesion directly to the dentino-enamel junction. The characteristic caries lesion is now evidenced in the area of the dentin immediately surrounding the terminal end of the lamella. More striking still is the evidence presented in Fig. VIII-A and VIII-B. There is only a slight lesion of the enamel, but a lamella extends from the periphery to the dentino-enamel junction; and again a characteristic yellowish-orange, identified with the carious process, is in evidence in the dentin immediately surrounding the dentin terminal end of the lamella.

Similar evidence is presented in Fig. IX, but with a more advanced state of dentin caries. The grenz-ray studies of these three specimens reveal that in Fig. VIII there is evidence of only slight decalcification, in Fig. IX there is no evidence of decalcification of the enamel, whereas in Fig. VII there is decalcification of that part of the enamel which in the black and white photomicrograph appears darkly pigmented.

An analysis of the evidence presented in Figs. I through IX reveals a series of progressive stages of caries, beginning with caries of the enamel alone and subsequently involving the dentin. Some of these enamel lesions are not associated with a lamella, and others are. Evidence clearly shows that in those cases of enamel caries which are not associated with a lamella, the caries must

extend through the entire depth of the enamel until it reaches the dentino-enamel junction, when the first evidence of dentin caries is discernible. In no case of enamel caries studied has there been any evidence of dentin caries if the enamel lesion did not extend to the dentino-enamel junction. As soon as the lamella is present, whether associated with an enamel lesion or not, as in Fig. IX, caries of the dentin, underlying the terminal ends of the lamella, is present. It must be remembered that in all the evidence presented (Figs. I through IX) there was no evidence of a clinical break in the enamel; and, with the exception of the specimens presented in Figs. VI and IX, no clinical means, not even x-ray, would reveal the presence of dentin caries, as it was revealed by the microscopic and grenz-ray studies.

It is evident that the enamel constitutes a slow path for caries development; whereas a lamella presents a direct and rapid path for the invasion of caries into the dentin. In all cases where a lamella is present, even one associated with an enamel lesion, caries is evident in dentin underlying the terminal ends of the lamella, although there is no evidence of any enamel disturbances in those areas directly surrounding the lamella. The lamellae traverse an area of normal enamel. As is well known, lamellae contain organic substance, are, in fact, organic tracts. The evidence tends to support the conclusion that the bacteria associated with the etiology of caries have an affinity with organic, rather than with inorganic, tissues and must therefore be regarded as proteolytic rather than acidogenic.

Structural Phenomena of Enamel.—In a previous study it was demonstrated that enamel forms in a matrix state before final maturation occurs.[20] It has also been demonstrated that organic enamel matrix contains as much as 28 to 33⅓ per cent of inorganic salts.[21] The maturation stage of enamel indicates a continuous increase of inorganic salts in the structure of the organic matrix. It is also established that the path of inclusion of the additional inorganic salts during the maturation stage is markedly different from the previously accepted belief. It does not follow the path of incremental formation of enamel, but begins at the most peripheral occlusal or incisal region of the crown after the full thickness of enamel matrix is established, and proceeds along a path in transverse relation to the incremental pattern. The formation and calcification stages of enamel development are therefore separated into two distinct processes.[20]

Based upon this knowledge, the possibility of a disturbance in maturation, as differentiated from hypoplasia, became apparent. Hypoplasia is a disturbance in the formation stage of the enamel matrix due directly to the disturbance of the ameloblasts. A gross hypoplastic lesion may, therefore, result in a definite deformity or a quantitative deficiency of structural formation in those areas of the enamel where the lesion occurs. It has been observed, however, that despite such structural deficiencies in the formation of the enamel matrix, the maturation stage of calcification may proceed normally.

A disturbance in the calcification process is known as hypocalcification. The enamel matrix may be normally formed but there may be a deficiency of

calcification in a localized area or there may be failure of calcification of the entire enamel. The enamel then remains in a matrix state.

It is pertinent to our discussion to note that it is *impossible by means of microscopic ground section study to differentiate between enamel in the matrix stage and fully calcified enamel.* The ground section studies of enamel affected by caries, therefore, do not reveal whether the affected parts of enamel were originally in an organic matrix state or in a state of complete calcification. Differentiation between enamel in a matrix state and the state of complete calcification can, however, be determined by microscopic studies of decalcified specimens of enamel, or by means of the grenz ray.

It is interesting in this connection to refer to the experimental studies of Mellanby.[22] Mellanby experimented with puppies using diets deficient in vitamins D and A and mineral salts. She described her findings on the enamel of the puppies' teeth as hypoplastic phenomena. Mellanby, at the time, was unable to differentiate between hypoplasia and hypocalcification. She showed her awareness that the character of the deficiency she observed was different from the usual hypoplastic phenomenon by differentiating it from the "gross hypoplasia (the type usually described in textbooks)." Her published evidence in the light of our present knowledge reveals, however, that in her decalcified sections she was dealing with the phenomenon of hypocalcification. In other words, the experimental deficiency diets Mellanby used resulted largely in a deficiency of the maturation stage of enamel. It is interesting, too, that as the result of her studies over a period of twenty years, Mellanby arrived at the conclusion that "extensive caries occurs in only 7 per cent of well-formed teeth," by which she means fully calcified enamel, wheras "extensive caries occurs in 74 per cent of teeth with fairly severe hypoplasia," by which she means hypocalcifications.[23]

Miller[3] and a host of observers have also concluded that the structural character of enamel is in some way related to the caries process. There is considerable evidence that localized areas of hypocalcification of enamel are fairly common in teeth of the mixed modern Europeans. Bodecker,[24] in decalcifying enamel under the microscope, demonstrated the presence of localized areas of enamel in an organic matrix state in teeth which in ground section study showed no differentiation between fully calcified enamel and enamel in an organic matrix state.

We believe that this phenomenon is an important factor in the understanding of the caries problem. While the evidence at present is not conclusive, there are sufficient indications that caries flourishes more on the soil of enamel in the state of organic matrix than on fully calcified enamel. The phenomena presented in this study show in ground section marked changes in localized areas of enamel. One would expect to find evidence of complete decalcification associated with such lesions, yet the grenz ray reveals the presence of considerable calcium salts. If the caries process were primarily an acid phenomenon the amount of inorganic salts present in these lesions would not be found. The behavior of the structure in these localized areas of enamel, on the other hand,

is quite similar to the reaction of enamel tissue in an organic matrix state when subjected to acid. The tissue immediately changes from an opaque white to increasingly deeper shades of brown, depending upon the concentration of the acid and the time during which the tissue is subjected to the acids. In this process the inorganic salts known to be contained in the matrix state of enamel are dissolved and the tissue gradually becomes softened, assuming a leathery consistency.

It appears to us that the phenomena described and the evidence presented in our photomicrographs is quite similar to, or characteristic of, the behavior of enamel in the state of organic matrix. It therefore strengthens the conviction that the caries process primarily subsists on organic tissue and secondarily results in decalcification.

Added to this evidence is the behavior of the disease associated with the presence of a lamella. The enamel in these cases remains virtually unaffected, the bacteria gaining rapid passage to the dentin through a direct organic path. The belief that the disease of caries is primarily proteolytic is therefore strengthened.

Attention must be called also to the observations by Buest and others[16-18] that in decalcification studies the enamel associated with caries is quite commonly found in an organic state. Explanations by these observers for this phenomenon are concerned either with the addition of protein material from saliva into the enamel, which increases the protein content of the enamel, or with the presence of bacteria, which are presumed to increase the organic content. The possibility or probability that the enamel at the site of caries was originally in a deficiency state of calcification of varying degrees becomes a very important consideration. We feel that this, therefore, is an important additional field for further investigation.

We have been discussing a phenomenon which we consider the mechanism of caries. It is apparent, largely from clinical studies, that there is one type of cavity formation which etiologically is acid. These cases evidence a destruction of the enamel surface, readily detected by x-ray and by clinical means. We now believe that this phenomenon occurs when enamel is in a fully calcified state. Once a pathway is established, the exposed dentin may become infected and the "true caries" process established.

The phenomenon of cavity formation which we have been discussing, and which we regard as "true caries," is primarily proteolytic in action, we believe. Clinically, if observed in the critical stage, the enamel, particularly of proximal surfaces, appears radiopaque by the x-ray, as if the proximal enamel were completely intact, whereas the dentin reveals a carious lesion. We have observed some of these cases clinically over a period of two or three years. While no clinical or x-ray change in enamel was observed, the caries phenomenon of the dentin progressed continuously.

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THE EFFECTS OF LARGE DOSES OF A VITAMIN D PREPARATION ON THE ORAL MUCOUS MEMBRANES OF HUMAN BEINGS*

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THIS report deals with a study of the oral reactions of a group of arthritic patients under treatment with Ertron‡ (a vitamin D preparation). It is the result of observations made at the Hospital for Special Surgery in New York as part of an extensive clinical investigation to determine the systemic changes occurring in arthritics after administration of this drug.

Dr. Sidney Jackson, the attending dental surgeon, made the clinical examinations. The complete histories of five cases were selected. These were found to be typical generally of a much larger number and are reported here in detail (Tables I and II). Twelve additional cases are also included.

During the entire experimental period only one medication was used (the vitamin D preparation).

GROSS EXAMINATION AND FINDINGS

On examination prior to treatment, the gums were found to be generally soft, spongy, edematous, tender, bleeding, and reddened especially around the necks of the teeth. In some instances the teeth were loose. The severity of the symptoms was somewhat less in those patients whose mouths gave evidence of hygienic care. But the unhealthy signs were present in varying degrees in all cases, even edentulous jaws showing spongy, flabby, and reddened tissues.

During the course of medication, Dr. Jackson describes the gingival condition as markedly improved in all instances. The gums were seen to be firmer, less prone to hemorrhage, normally pink in color, and not excessively tender. In some patients, loose teeth returned to normal fixity.

The second phase of the investigation consisted of a histologic study of the seventeen patients under observation. Whereas many forms of treatment had been used previously, medication during the experimental period was restricted to the vitamin D preparation taken orally in capsule form. Each capsule contained 50,000 U.S.P. units. The dosage varied from 3 to 6 capsules (150,000 to 300,000 units) daily. (Tables I, II, and III.) Gingival biopsies were made

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‡Ertron is described by Horwitz and Joseph[1] as a mixture of unactivated ergosterol, calciferol, and fairly large proportions of a vitamer D§ which is not identifiable at present with any of the vitamers previously characterized by specific properties. . . .

§The term "vitamer" is used to differentiate individual members of a group of vitamins having a common action.

TABLE I. BLOOD ANALYSES OF FIVE PATIENTS RECEIVING LARGE DOSES OF A VITAMIN D PREPARATION BEFORE AND AFTER TREATMENT PERIOD*

	CASE 1 H. P., MALE		CASE 2 M. P., FEMALE		CASE 3 S. P., FEMALE		CASE 4 M. M., FEMALE		CASE 5 H. G., FEMALE	
Age (years)	23		46		37		34		54	
Complaint	Pain in back and groin		Generalized pain in joints		Pain in wrists, hands, knees, and ankles		Pain in joints, back, legs, and arms		Pain in head, neck, arms, back, legs	
Duration (years)	3		13		2½		5		2	
Diagnosis	Arthritis		Rheumatoid arthritis		Rheumatoid arthritis		Menopausal arthralgia		Osteoarthritis	
Ertron dosage†	13,200,000 units in 86 days		60,900,000 units in 210 days		27,300,000 units in 147 days		29,500,000 units in 140 days		10,500,000 units in 63 days	
*Blood Tests at Beginning and End of Treatment Periods**										
Calcium	11.0	10.0	9.8	9.8	10.3	10.4	10.9	12.3	10.3	13.3
Phosphorus	2.56	2.45	3.09	2.91	3.25	2.80	2.68	2.88	3.85	3.25
Phophatase (B.U.)	4.26	4.53	3.56	3.36	9.07	8.63	3.33	2.16	1.60	2.39
Fasting sugar	93	98	93	88	100	93	107	79	100	100
Uric acid	3.4	4.7	4.2	4.2	4.2	4.7	2.7	4.7	2.1	3.4
N. P. N.	30	32	40	30	29	33.3	30	37.5	27	30
Total protein	7.82%	7.00%	6.01%	6.70%	6.95%	7.30%	7.79%	7.53%	7.44%	6.77%
Sedimentation rate (1 hr.) (Westergren method)	15 mm.	23 mm.	8 mm.	10 mm.	23 mm.	34 mm.	8 mm.	24 mm.	16 mm.	20 mm.
Hemoglobin (Sahli)	94%	100%	85%	82%	70%	82%	90%	85%	90%	86%
R. B. C.	4,880,000	4,950,000	4,400,000	4,400,000	4,040,000	4,080,000	4,610,000	4,620,000	4,580,000	4,480,000
W. B. C.	7,900	11,100	6,000	7,400	7,000	8,500	4,100	6,700	5,000	6,000
Polymorphonuclears	68%	52%	74%	64%	57%	63%	35%	45%	62%	60%
Lymphocytes	31%	46%	22%	34%	42%	37%	65%	55%	36%	40%
Mononuclears		2%								
Monocytes	1%								2%	
Kline test	Negative		Negative		Negative		Negative		Negative	

*From the hospital case histories.
†The vitamin D preparation.

TABLE II. GINGIVAL BIOPSY FINDINGS BEFORE AND AFTER TREATMENT PERIOD

ERTRON DOSAGE	CASE 1 H. P., MALE 13,200,000 UNITS IN 86 DAYS		CASE 2 M. P., FEMALE 60,900,000 UNITS IN 210 DAYS		CASE 3 S. P., FEMALE 27,300,000 UNITS IN 147 DAYS		CASE 4 M. M., FEMALE 29,500,000 UNITS IN 140 DAYS		CASE 5 H. G., FEMALE 10,500,000 UNITS IN 63 DAYS	
Epithelial quality	+++	++++	++	+++	++	+++	++	++++	++	++++
Keratin	+++	++++	+++	+++	+	+	++	++++	++	++
Hyperplasia	−	++	−	++	−	++	−	+++	++	++++
Vacuolization	+	+	−	−	−	−	++	++	+	+
Pearls	−	−	−	−	−	−	++	++	+	+
Connective tissue edema	++	−	+	+	+++	+++	−	−	++	+
Fibroblasts	+	++	+	++	+	+++	+	+++	−	++
Fibrocytes	+++	+++	+++	+++	++++	++	++++	+++	++++	+++
Vascularity	++	+++	+	+	++	++	++	+++	++	++
Collagen	+++	+++	++	+++	+++	++++	+++	++++	+++	++++
Inflammation	+++	++	+	+	+++	+++	++	++	++	+

before treatment was begun and again in the same area after a lapse of one to ten months. The examination of these tissues constitutes the basis for this report.

Blood count, sedimentation rate (Westergren method), and blood chemistry were taken at the beginning and at the end of the experimental period. Urinanalyses were also done before and after. These tests were for color, reaction, specific gravity, albumin, sugar, acetone, white blood cells, and epithelial cells. The Kline test was a routine procedure.

The gingivae were sectioned in the usual manner. Hematoxylin and eosin, and Masson's trichrome stains were used. (Tables II and III.)

RESULTS

The findings in urinanalyses, the Kline tests, and the blood counts were essentially negative. An increase in the sedimentation rate in the final test was noted, and the data on five cases are tabulated in Table I. If 20 is taken as the figure representing the upper limit of normal, it may be said that the sedimentation rate in two cases rose to slightly above normal; two remained within normal limits; and the rate in one case was above normal at both testings. Another deviation was seen in the elevation of the blood calcium in two cases and its reduction in one. In two, this value remained unchanged.

Microscopic Examination.—The final biopsies showed improvement in the quality of the epithelium as judged by staining reaction and general appearance. In two cases there was an actual increase in the thickness of the keratin layer (Figs. 2, 4, and 8). In all cases the keratin seemed more mature, not as prone to fragmentation (Figs. 5, 7, and 9) during sectioning, and lacked its former frayed appearance. Generally, it resembled the keratin of the skin, which has fewer flattened nuclei than are ordinarily seen in the gingival mucosae. Hyperplasia (Figs. 6 and 10) of both the epithelium and connective tissue was seen in all but two instances, being extreme in one case, moderate in ten, and slight but definite in the remainder. Epithelial pearls were noted in the

TABLE III. GINGIVAL BIOPSY FINDINGS AFTER TREATMENT PERIOD

CASE NUMBER	SEX	AGE	DIAGNOSIS OF TYPE OF ARTHRITIS	DOSAGE U.S.P. UNITS	DAYS	EPITHELIUM QUALITY	KERATIN THICKNESS AND QUALITY	PEARLS	HYPERPLASIA	FIBROBLASTS	VASCULARITY	INFLAMMATORY EXUDATE	PERIVASCULAR INFILTRATION IN MUCOSA	INTERDENTAL PAPILLA—UPPER*
6 C. F.	♀	35	Rheumatoid	4,200,000	28	++	++	++	±	++	+++	++	+	L2
7 J. W.	♀	39	Mixed	13,300,000	119	+++	+++	0	+++	+	+	0	0	L2
8 H. F.	♀	46	Not determined	13,600,000	63	+++	++	0	++	++	+++	–	0	L2
9 F. S.	♀	43	Rheumatoid	16,250,000	71	+++	+++	±	+++	+++	+++	0	0	L3
10 K. M.	♀	39	Mixed	36,400,000	182	+	++	+	+++	+++	+++	–	+	L1
11 M. L.	♀	37	Chronic rheumatoid	37,800,000	140	++	+++ +++	+	+++	++	+++	0	+	R2 Edentulous
12 H. D.	♀	68	Osteoarthritis	37,800,000	147	±	+	0	+	+	+	++	++	L3
13 R. B.	♀	39	Rheumatoid	58,800,000	308	+++	++++	±	+++	++	+++	0	0	R2
14 R. H.	♂	57	Rheumatoid	61,400,000	252	+++	++++	0	+++	+++	++++	0	+	L2
15 P. C.	♂	68	Chronic rheumatoid	79,800,000	238	+++	++++	0	+++	+++	+++	0	+	R2
16 M. R.	♀	30	Rheumatoid	91,700,000	140	++	+++	+	+++	+++	+++	±	+	L1
17 M. H.	♀	44	Osteoarthritis	116,200,000	287	+++	+++	0	+++	++	++++	0	+	R3

*L = left. R = right.

Fig. 1.

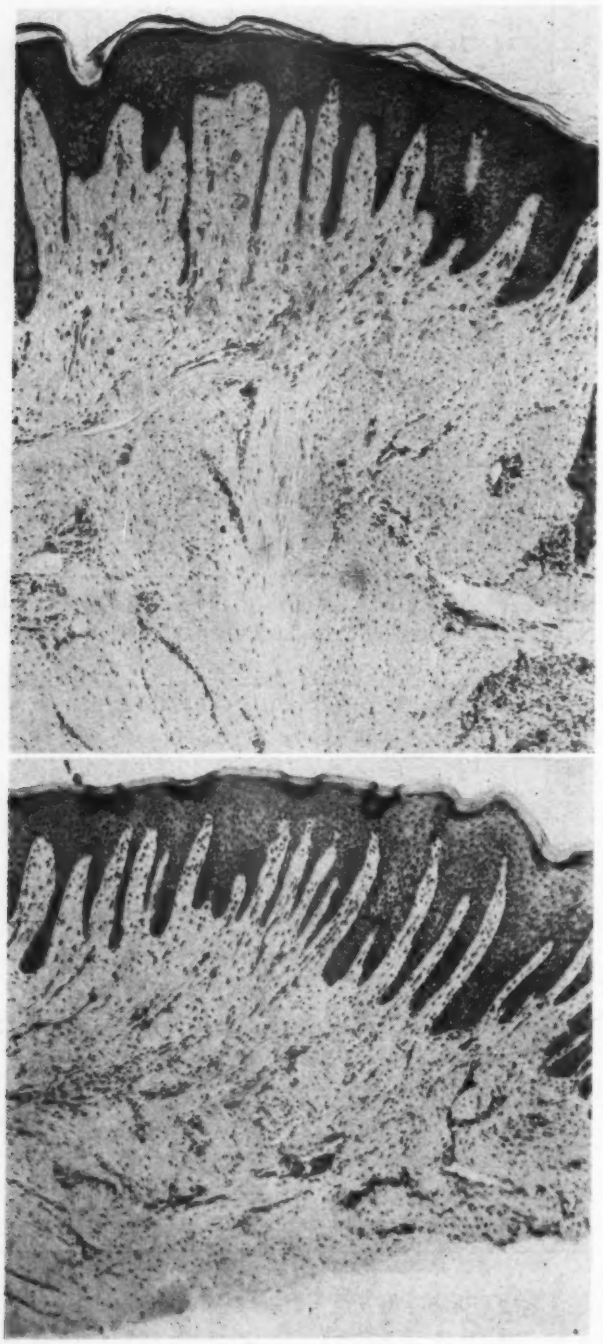


Fig. 2.

Fig. 1.—Case 1. H. P., male. Pre-experimental. Upper right lateral incisor interdental papilla; alveolar gingiva. The keratin layer is present but is fragmented and has been stripped loose during sectioning. The prickle-cell layer is not very wide; the pegs are pointed. Corium: the connective tissue layer is much thicker than normal and is made up almost entirely of fibrocytes. There are many perivascular foci of inflammation with some edema. (×80.)

Fig. 2.—Case 1. After treatment period. Upper left cuspid interdental papilla; alveolar gingiva. The general appearance of this section of gum is much healthier and gives the impression of being much firmer. The keratin layer is uniformly thick and is adherent to the underlying tissue. It did not strip off during sectioning as it did in the pre-experimental period. The prickel-cell layer is hyperplastic; the epithelial pegs present a more uniform pattern. Corium: the connective tissue layer is not as thick as in the pre-experimental period. It is composed primarily of fibroblasts, being more cellular in structure. There are markedly less perivascular foci of inflammation. Numerous small capillaries are in evidence, suggesting increased blood supply without vascular congestion. (×80.)

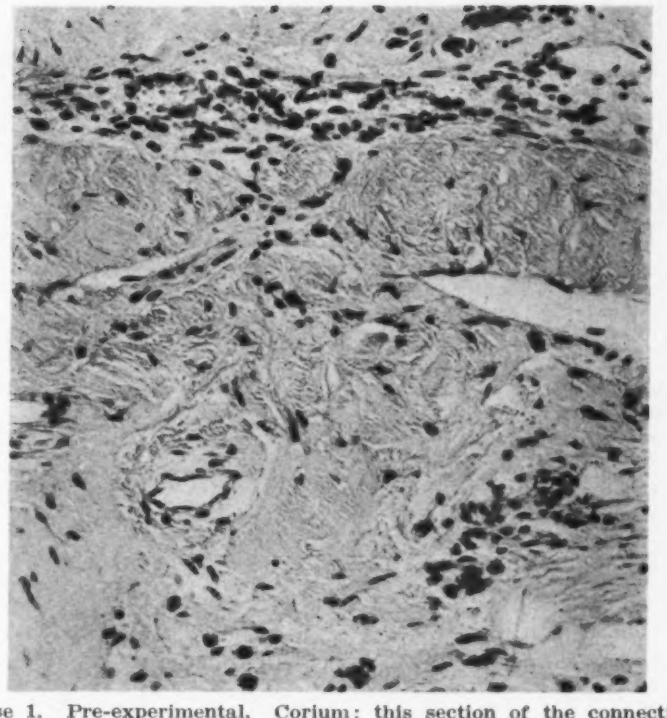


Fig. 3.—Case 1. Pre-experimental. Corium: this section of the connective tissue layer shows the presence of marked edema, which may account for the increased thickness of the corium. The collagen bundles are fragmented and appear of poor quality. The foci of perivascular infiltration are numerous. There are numerous distended blood vessels which probably account for the congested red color of the gum. (×336.)

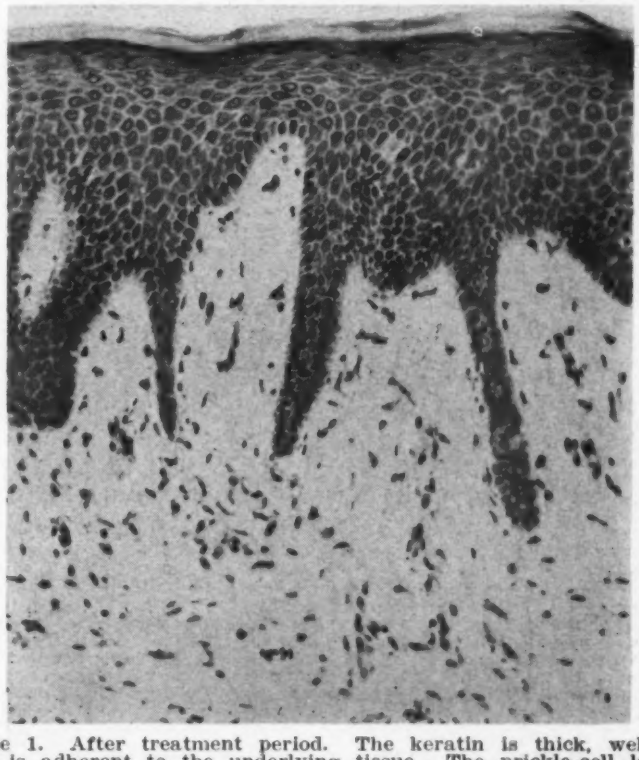


Fig. 4.—Case 1. After treatment period. The keratin is thick, well-defined, mature in character, and is adherent to the underlying tissue. The prickle-cell layer is firm and well formed. There is some suggestion of pearl formation. Corium: the connective tissue is more homogeneous (not fragmented bundles), is more cellular, being composed primarily of fibroblasts with markedly less inflammatory foci. The whole section gives the appearance of a normal healthy firm gum. (×280.)

Fig. 5.

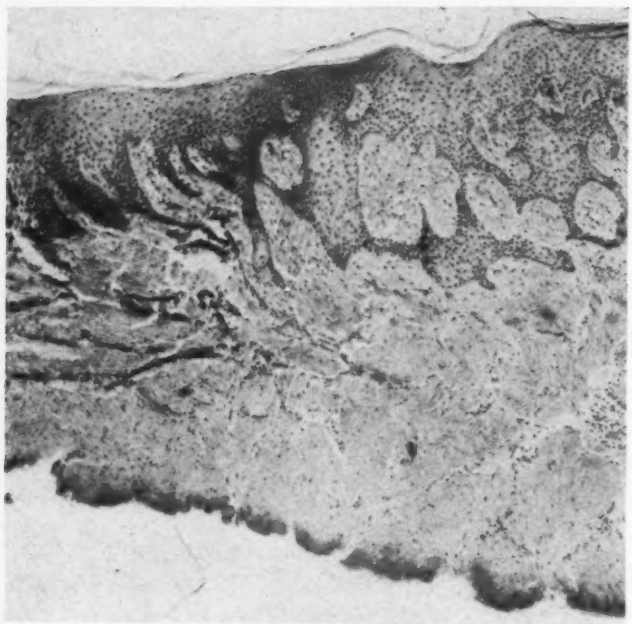


Fig. 6.

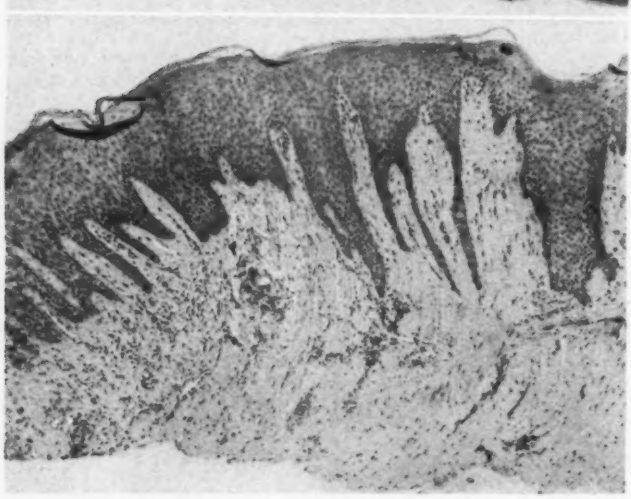


Fig. 5.—Case 4. M. M., female. Pre-experimental. Upper central incisor interdental papilla. Alveolar gingiva. The keratin layer is very thin, fragmented, of poor quality, and is almost entirely stripped off. The prickle-cell layer shows slight hyperplasia with some elongation of the epithelial pegs. There is a suggestion of epithelial pearls. Corium: the connective tissue layer is much wider than normal and is composed of dense collagenous bundles, this layer being composed almost entirely of fibrocytes. Many foci of perivascular infiltration are in evidence. (×80.)

Fig. 6.—Case 4. After treatment period. Upper right lateral incisor interdental papilla. Alveolar gingiva. There is a marked change in the keratin layer as compared to the pre-experimental section. It is of better quality, thicker, adherent to the underlying tissue and there is some parakeratosis near the tip. The epithelial layer is, as a whole, more hyperplastic. Numerous epithelial pearls are in evidence. The epithelial pegs are more uniform in length and structure. Corium: the connective tissue is of better quality, is more cellular and is composed primarily of fibroblasts. More capillaries are present but there is an absence of edema or distention of the blood vessels. The whole tissue seems firmer and more normal than the pre-experimental section. (×80.)

alveolar gingivae of eight patients, all female. Connective tissue edema was not materially affected. A significant finding was the increased number of fibroblasts present at the termination of treatment (Fig. 12). The capillary bed was extremely prominent in two cases and moderately so in ten. Inflammatory exudate (Figs. 1 and 3) at the crest of the interdental papillae was absent in seven cases and present in eight at a higher level in the mucosa (Figs. 11 and 12). The outstanding changes in the gingival tissues as a result of the experimental procedure may be summarized as follows: the tissues were of improved quality as evidenced by staining reaction; the keratin was increased in amount and healthier in appearance; hyperplasia was apparent by the presence of more numerous nuclei in both the epithelium and the connective tissue, and the greater width of the epithelium; a larger number of fibroblasts was seen; and hyperemia was manifest by the increase in prominence of the capillary bed.

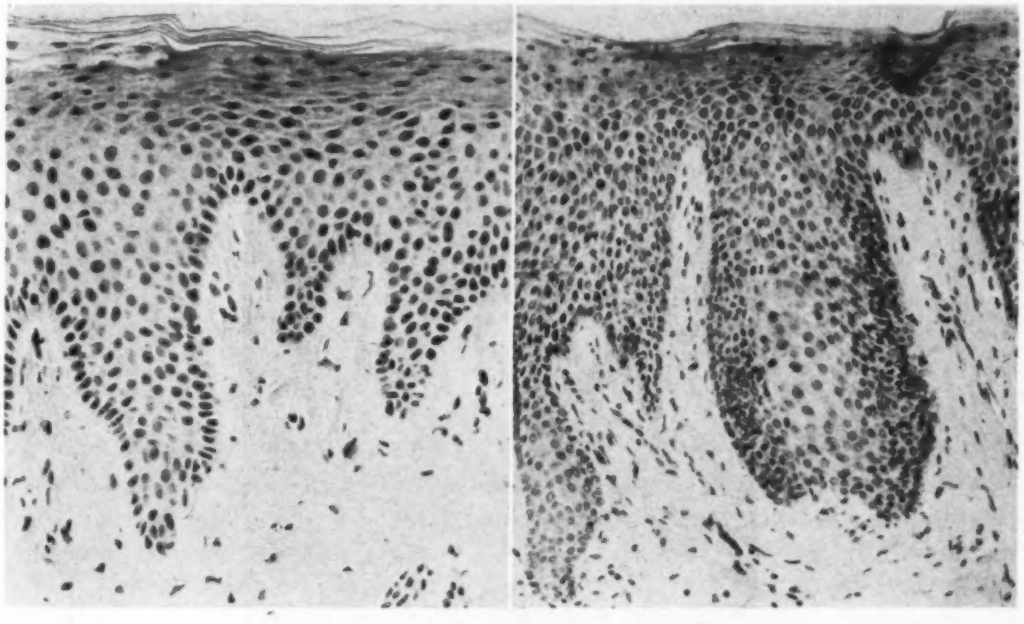


Fig. 7. Fig. 8.

Fig. 7.—Case 4. High power of Fig. 5. (×378.)
Fig. 8.—Case 4. High power of Fig. 6. (×245.)

The quantitative changes in the tissues were slight; the qualitative changes were marked. There was no apparent proportional relationship between dosage of the vitamin D preparation and any one of the findings listed above.

In a recent study of the effects of large doses of vitamin D on the teeth and jaws of rats and on human beings, we found a resultant stimulative reaction.[2] Other experiments of dental interest with vitamin D are reported in the literature, but they are also concerned chiefly with the hard structures of the oral cavity. The present study deals exclusively with the oral mucosa.

The findings disclose both clinical and histologic evidences of improvement in the gingivae, and our interest centers around the possible mechanism by which this improvement was brought about.

Fig. 9.

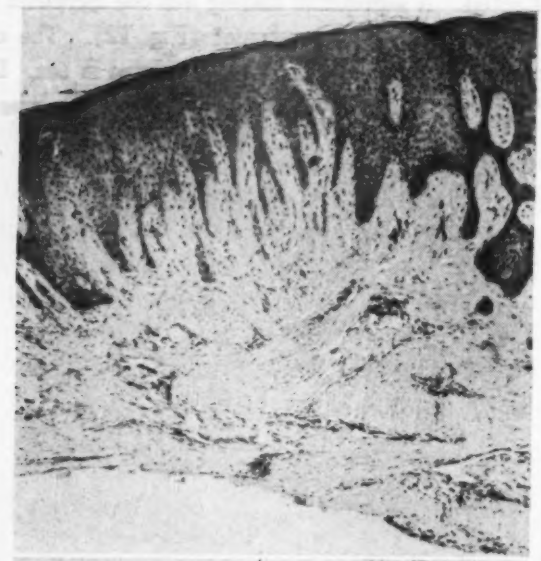


Fig. 10.

Fig. 9.—Case 5. H. G., female. Pre-experimental. Upper left lateral incisor interdental papilla; alveolar gingiva. The keratin layer is very thin, fragmented, and has stripped off during sectioning. There is some intercellular edema in the prickle-cell layer with slight evidence of hyperplasia. Corium: Consists of dense collagenous bundles and is composed primarily of fibrocytes. This connective tissue layer is not larger than normal. There is some edema in the corium with numerous perivascular foci of infiltration. (×80.)

Fig. 10.—Case 5. After treatment period. Upper right lateral incisor interdental papilla; alveolar gingiva. The keratin layer is only slightly increased in thickness but it is definitely of better quality. There is marked hyperplasia of the prickle-cell layer and no edema is present. There is a suggestion of pearl formation. Corium: the connective tissue is of better quality, is firmer, is much more cellular and with a preponderance of fibroblasts. There is an absence of edema and definitely less perivascular foci of infiltration. (×80.)

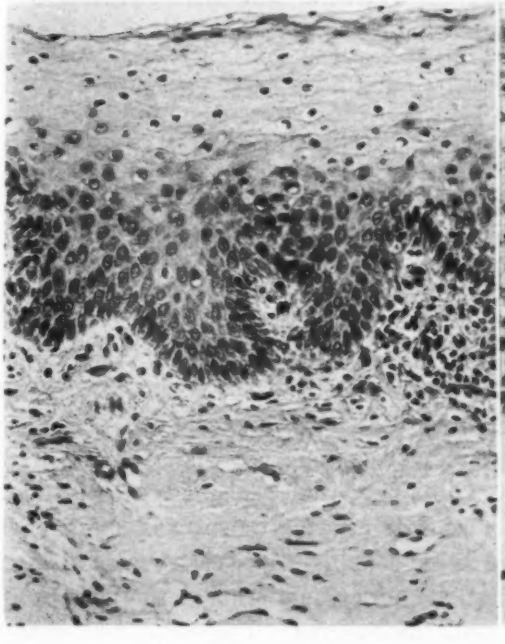


Fig. 11.

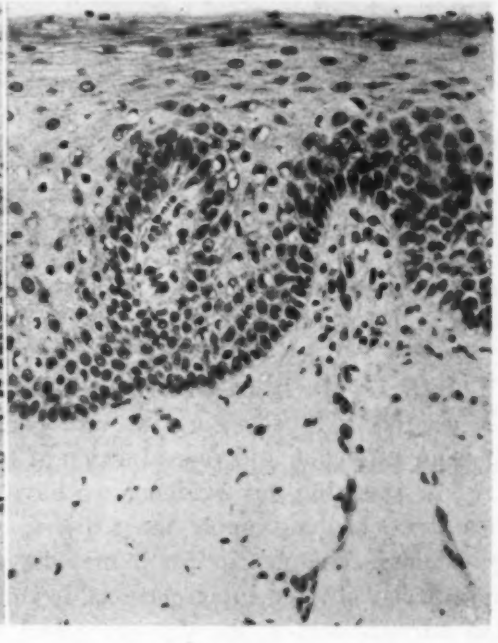


Fig. 12.

Fig. 11.—Case 5. Pre-experimental. Upper left lateral incisor papilla; mucous membrane. No keratin is normally present in this area and none is seen in this section. The epithelial layer shows marked edema. Corium: marked perivascular foci of inflammation and edema is seen throughout the corium. (×315.)

Fig. 12.—Case 5. After treatment period. Upper right lateral incisor papilla; mucous membrane. There is no keratin layer present (normal for this area). There is definite evidence of hyperplasia of the epithelial layer. Corium: the connective tissue is more homogeneous, consisting primarily of fibroblasts. Perivascular infiltration is reduced. No evidence of edema is seen. This section seems firmer, more nearly normal than the pre-experimental section. (×343.)

That the vitamin D preparation used here has a systemic effect has been indicated in other studies. The dental application of such response is demonstrated here. However, since there was no previous evidence of a vitamin D deficiency, the effect may be nonspecific. The action may be one of stimulation to the general health of the gingivae as seen in the extraprominence of the capillary bed of the papillae. This hypothesis may be applied further to the increased blood supply, which in turn may be responsible for the hyperplasia noted microscopically and for the better quality of the tissues, resulting in a denser keratin layer.

My thanks to Miss Marie Petrilli of the Hospital for Special Surgery for assistance in obtaining information from the hospital records.

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PREGNANCY GINGIVITIS: HISTORY, CLASSIFICATION, ETIOLOGY

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INTRODUCTION

THIS report is based on a clinical and microscopic study of the changes occurring in the gingivae and oral mucous membranes during pregnancy. It is an extension and re-evaluation of previous reports.

In assessing our evidence we have extensively reviewed medical and dental literature bearing upon the subject of endocrine relationships. The general physiologic changes in pregnancy have been considered, stressing some of the specifically studied interrelationships of such endocrine glands as the hypophysis, the adrenal cortex, the thyroid gland, and the ovaries. The histologic changes that glandular imbalances may produce in the gingivae have been particularly noted.

A clinical classification of pregnancy gingivitis is included. Five major classifications are distinguished, ranging from the commonest form, characterized by bleeding of the gums, to the so-called "pregnancy tumor."

HISTORY

The fact that pregnancy has profound effects on the gingival tissues has been known for a long time, long before the endocrine changes in pregnancy were known or endocrine effects on the oral tissues even suspected. Thus, Oakley Coles, in 1874, described fairly accurately the condition which has subsequently been called pregnancy gingivitis. However, Coles believed that this condition was purely coincidental with pregnancy and says, "Whilst therefore speaking of those changes that take place in the gums, we must bear in mind their appearance is due to influences that are in operation at other times than during pregnancy and that beyond the fact of their coincidence, they would call for no special attention."

However, in 1877, Drs. A. and D. Pinard stated that the gingivitis was induced by pregnancy although the proximate cause was not known. Clinically, they used a solution of iodine, glycerol, tannin, and potassium chlorate with good effect. At the close of the nineteenth century this disturbance was evidently fairly well accepted, since the condition of pregnancy gingivitis and palliative treatment is included in the fifth edition of *A System of Oral Surgery* by Garretson.

An interesting case history was given by Zentler in 1899. He describes recurrent gingivitis during three pregnancies. In each pregnancy, there was some hypertrophy of the gums which was more pronounced and required a greater recovery period in the third pregnancy. The condition persisted in the latter pregnancies despite careful treatment of the mouth; it disappeared after parturition without any local interference in the first two pregnancies, and with very little interference following the third pregnancy.

Talbot (1913) stated that interstitial gingivitis was always present in pregnancy to a more or less marked degree. He believed that the condition was due to autointoxication caused by retained toxins of the mother and fetus.

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Although, as has been shown, the condition of pregnancy gingivitis has been known for a long time, little progress was made in defining the etiology. Thus, Monash (1926), reporting his observations on six cases of pregnancy gingivitis, stated that the condition was entirely inflammatory in nature and merely represented an exaggerated degree of generalized gingivitis. The treatment suggested consisted of thorough cleaning and scaling of the teeth, removal of badly decayed teeth and poor dental restorations, massage of the gums, and instruction in the proper use of the toothbrush. If necessary, any growth should be excised after parturition. As for etiology, little was said except that there was some gingivitis before pregnancy which was intensified during pregnancy. Monash agreed with Coles that there is no gingivitis peculiar to pregnancy.

Merritt (1930) believed that there was no gingivitis peculiar to, and found only in, pregnancy. However, gingivitis accompanied by oral sepsis is found during pregnancy. There are hypertrophic growths which increase in size during pregnancy and then disappear after delivery.

A study of sixteen cases of pregnancy tumors was made by Blum in 1931. The tumors occurred in various months during pregnancy, and in women of varying parity. For treatment, Blum stated that the tumor should be removed surgically to prevent recurrence, especially since the operation is simple and conservative. Apparently, pregnancy tumors are blood tumors originating from vasomotor disturbances of the endothelial layer of blood vessels. However, Blum seriously considered a disturbance of the endocrine balance as an etiological factor, but stated that there was no proof that the internal secretions were involved, or if so, to what extent.

Hirschfeld (1932) stated that pregnancy manifests itself through the gums in several ways, namely, marginal gingivitis, hypertrophy of the gums, especially of the interdental papilla, and proliferations from under the gum either interproximally or labiolingually. The condition may be generalized, or especially marked where there is fool impaction, traumatic occlusion, lack of function, or any other traumatizing factor. The hypertrophied masses may continue to grow until after parturition when they usually disappear.

The earliest work to correlate the endocrines and the condition of pregnancy gingivitis was made in 1933, when Ziskin, Blackberg, and Stout made an experimental study and histopathologic interpretation of the gingivae during pregnancy. They found some form of pregnancy gingivitis in 70 per cent of the patients examined, thus confirming the work of Freud, Pinard, and Schmidt (1932) (cited by Schour, 1934) who found gingivitis in from 53 to 60 per cent of pregnant women, whereas control studies in nonpregnant women showed 15 to 18 per cent incidence. As a result of their clinical and histologic observations, and experimentally induced gingivitis in monkeys simulating pregnancy gingivitis, Ziskin, Blackberg, and Stout concluded that the significant change in the gums in pregnancy gingivitis is a hyperplasia of the epithelium and a loss of surface keratin, with the probability that hormones are the causative agents. The so-called pregnancy gingivitis was renamed more generally as "hormonal gingivitis" by Ziskin (1936).

METHODS

A series of biopsies were taken at different months during gestation from different pregnant women, who had clinical manifestations of pregnancy gingivitis. Microscopic studies of twenty of these cases are listed here. In the epidermis, we looked for the presence or absence of surface keratin and stratum granulosum, edema and hydropic degeneration of the stratum spinosum, hyperplasia of the stratum germinativum, the type of peg pattern, and the number of mitotic figures per section. In the lamina propria, we examined the sections for inflammatory cell infiltration, hyperemia and condition of blood vessels, and the relative amounts of collagen and fibrous connective tissue. Clinical photographs were also taken and are included. Results are listed in Table I.

TABLE I. GINGIVAL CHANGES DURING PREGNANCY

			EPIDERMIS							LAMINA PROPRIA			
	PATIENT'S INITIALS	MONTH OF PREGNANCY	STRATUM CORNEUM	STRATUM GRANULOSUM	HYDROPIC DEGEN. IN ST. SPINOSUM	HYPERPLASIA IN STRATUM GERMINATIVUM	PEG ELONGATION	MITOSIS	PEARL FORMATION	VESSEL DILATION	COLLAGEN	FIBROUS CONNECTIVE TISSUE	INFLAMMATORY CELLS
1	A. H.	4	+	±	+	-	+	0	0	++	+	+	-
2	V. H.	5	±	-	+++	+	++	0	0	+	+	++	+
3	A. K.	5	±	+	+	±	++	0	0	++	+	+	++
4	E. B.	6	-	-	++	±	+++	0	0	++	++	+	++
5	W. L.	6	±	-	++	-	++	0	±	++	+	+	+
6	L. G.	7	±	-	++	+	++	0	0	++	+	+	++
7	Y. G.	7	±	±	+	+	++	0	0	++	+	+	++
8	M. R.	7	±	±	+	-	++	0	0	±	++	++	±
9	G. S.	7	++	+	±	+	++	0	+	-	++	++	-
10	E. Y.	7	±	+	++	+	+	0	0	+	+	+	+++
11	H. G.	8	-	-	++	+	+++	0	0	+	+	+	+++
12	S. K.	8	-	-	++	+	++	0	0	-	+	++	+++
13	F. N.	8	-	-	+	-	++	0	+	+	++	+	++
14	W. P.	8	-	-	++	+	+	0	0	+	++	++	+++
15	B. E.	9	-	-	++	-	++	0	++	±	++	++	+++
16	E. F.	9	±	-	±	+	++	±	0	++	+	+	++
17	P. G.	9	±	+	+	+	+	0	0	+	++	+	+++
18	M. C. M.	9	-	±	++	-	++	0	0	++	+	+	+++
19	I. K.	9	±	±	+	±	++	0	0	+	+	+	++
20	D. L.	9	±	+	-	+	++	±	0	-	+++	++	-

Key: O = absent. - to +++ = degree of change.

The most significant changes were: Disappearance of the stratum corneum as pregnancy advanced from the fourth to the ninth month, and its reappearance during the ninth month; hydropic degeneration; hyperplasia; epithelial peg elongation; inflammatory reaction in the lamina propria.

A series of biopsies were also taken from eight patients having some evidence of an endocrine disorder and from mucous membranes of three rhesus monkeys treated experimentally with hormones. These are included for comparison with the pregnancy group. Photomicrographs and clinical photographs of representative cases are also included.

RESULTS

A. *Clinical Classification of Pregnancy Gingivitis.*—A clinical classification of pregnancy gingivitis is presented below, which is based on several hundred cases examined (but not included in this report). The classification represents the progressive changes in pregnancy, and, therefore, more than one type may be present in the same case. There is no correlation between the microscopic changes and the clinical classification since the microscopic changes are essentially the same in all classes of pregnancy gingivitis, except for particular cases in which the microscopic changes deviate from the usual findings.

Class I: The first form of pregnancy gingivitis, and by far the most common, is characterized by bleeding of the gums. On questioning the patient, it may be ascertained that the tendency for the gums to bleed when traumatized by the toothbrush, food, etc., developed after the onset of pregnancy; or, if the symptoms were present before pregnancy, it became exaggerated after conception. Hemorrhage is also easily induced by probing.

Class II: The next recognizable form is a slight change in the interdental papillae. The papilla loses its stippled appearance, becoming glossy or "glassy" looking. Its color changes from pink to old rose. There is some edema, creating a mildly puffed appearance. Because of the swelling, the papilla no longer maintains the sharply pointed architecture of a pyramid, but becomes somewhat blunted at the tip. The symptoms are seen early, and if uncomplicated by local conditions, may persist in an unexaggerated state throughout the term. After parturition the gums generally return to normal without treatment. It should be pointed out here that the changes just described may be so slight as to be easily overlooked unless careful examination is made. On the other hand, they may be gross enough to be readily discernible. (Fig. 1.)

Class III: In the third type of pregnancy gingivitis, usually only the free gum margin is involved. It takes on the color and appearance of a raspberry. The highly inflamed tissue bleeds easily on being probed. Its most frequent site is the anterior gums, although it may be found in other locations. This form is designated "raspberry red gums." (Fig. 2.)

Class IVA: The fourth form, a generalized hypertrophy of the tissue, is designated "hypertrophic gingivitis of pregnancy." The interdental papillae become noticeably enlarged, losing their natural contours. The color may vary from an old rose to a deeper, cyanotic hue. In the presence of irritation, the borders may become bright red. The amount of involvement varies. One, a few, or many papillae may be involved in one or more sections of the mouth. The tumefaction of the individual papilla is gross in some and slight in others. (Fig. 3.)

Class IVB: The enlargement of the papillae is due in part to edema, and also to hyperplasia of the epithelium. Sometimes, a proliferation tissue forms. As it grows, the normal gum is pushed back, a straight blanched line of demarcation resulting. In some instances, the proliferation tissue, which emerges from the undersurface of the papillae, may grow over and cover in part the crown portion of the teeth. Here, then, is created a favorable site for mouth sordes to collect and act as an irritant, additional proliferation resulting. Other traumatic conditions, such as food impactions, the presence of calculus, overhanging margins of previously placed prostheses or sharp margins of cavities, and lack of function in a small area, further complicate the pathologic process. However, sometimes no irritants whatever are apparent. (Fig. 4.)

Class IVC: In a few cases that we have observed, several of the interdental papillae had grown to such proportions that they took on the appearance of epulides. As the individual papilla spreads laterally, the attachment to the gingiva becomes narrowed, giving the appearance of a pedunculated tumor. But even in this distorted stage, the tumors must still be classed as indicative of hypertrophic gingivitis, since they respond to treatment for this group rather than for epulis. (Fig. 5.)

Class IVD: In other cases, the proliferation starts from the undersurface of the free gum margin and spreads along the gingival crevice, covering the tooth for a few millimeters without involvement of the interdental papilla. (Figs. 6, 7, 8, 9, and 10.)

Fig. 1.



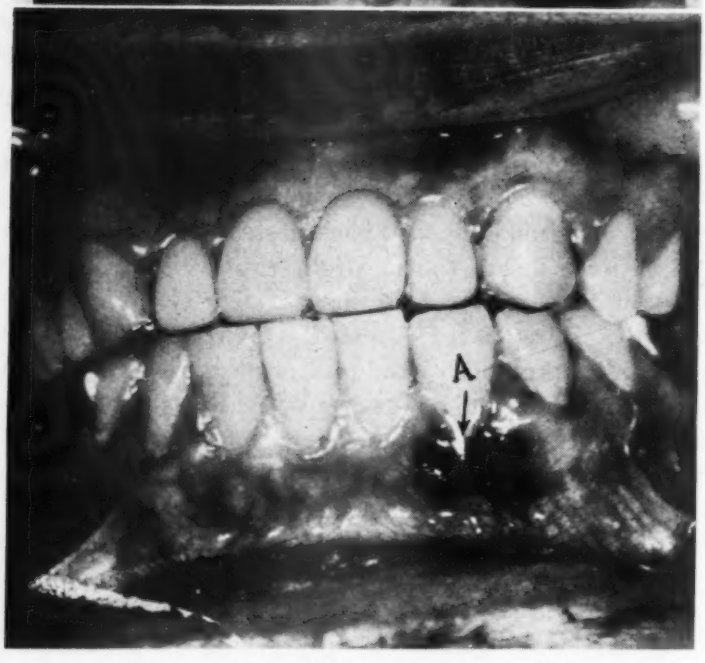


Fig. 2.

Fig. 1.—S. K., aged 24 years, white, multipara (third pregnancy), eighth month. Class II. Shows the mildly edematous appearance of the interdental papillae with blunted rather than pointed crests. They are glassy or shiny in appearance and of an old-rose color.

Fig. 2.—C. M., aged 29 years, white, multipara (second pregnancy), eighth month. Class III at *A* shows highly inflamed and vascular gingivae. The color and appearance are that of a raspberry.

Fig. 3.





Fig. 4.

Fig. 3 .—C. D., aged 20 years, Negro woman, primipara, seventh month. Class IVA. Shows generalized hypertrophy of the gingivae. The tumefaction of the interdental papillae is most marked in the lower incisor region. The gingival color varies from an old rose to a deeper cyanotic hue.

Fig. 4.—A. K., aged 23 years, white, primipara, fifth month. Class IVB. Shows proliferation tissue emerging from the undersurfaces of the papillae between the upper left lateral and central incisors, and the lateral incisor and cuspid. There is a distinct line of demarcation between the normal gingivae and the proliferation tissue. An early stage of this change is seen in the lower anterior interdental papillae.

Fig. 5.



Fig. 6.

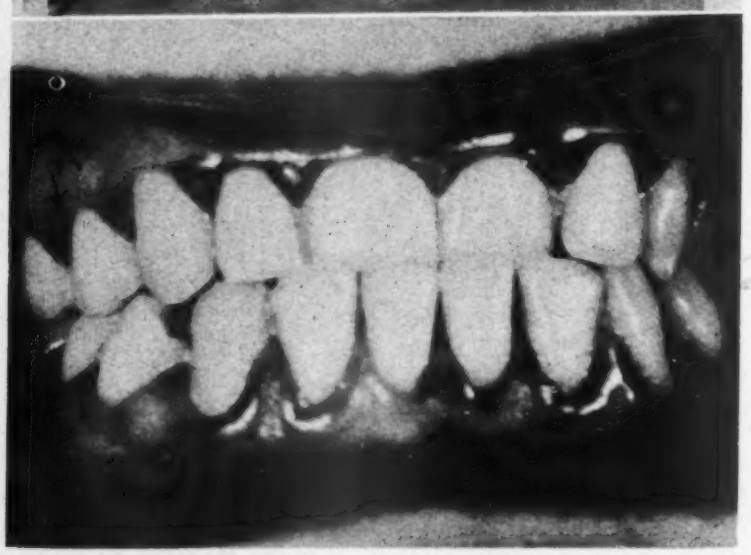


Fig. 5.—J, B., aged 26 years, white, multipara, fifth month. Class IVC. Shows overgrowth of papilla between upper left cuspid and premolar to such an extent that it looks like an epulis.

Fig. 6.—H. C., white, multipara, sixth month. Class IVD. Lower anterior labial gingivae show proliferation from undersurface of free gum margin without involving interdental papillae. The new proliferation tissue has pushed aside the normal gingiva, the line of demarcation being seen at *A* as a blanched line. The new granulation tissue in both upper and lower gingivae is pigmented. Several years following pregnancy the patient was re-examined and the pigmentation still persisted. (An increased deposition of pigment in the nipples, vulva, linea alba, navel, and face is found. Pigmentation on the face, when marked, may appear as a mask and the condition has been called cloasma gravidarum [the mask of pregnancy]. [Davis, Boynton, Ferguson, and Rothman, 1945]).

Class IVE: Another type of change in the same category, which occurs less frequently, is the formation of a pseudopapilla. Here, the interdental papilla may not be enlarged, and the pseudopapilla may be the same color as the rest of the gingivae. However, a definite horizontal line of separation, forming a cleft, can be seen at the base of the papilla. If a flat blunt blade is placed in this line, it is a simple procedure to lift the growth from the normal gum, and, with little effort, it can be removed. (Fig. 11.)

In all instances of hypertrophic gingivitis of pregnancy, the gums bleed easily on being probed, but are seldom, if ever, painful.

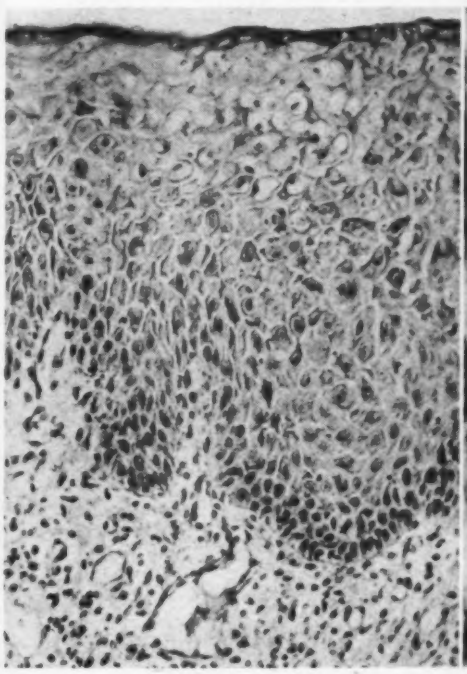


Fig. 7.

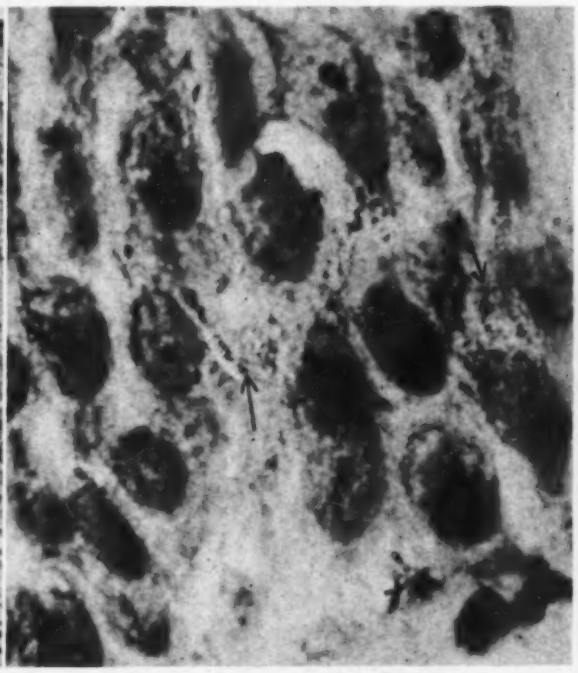


Fig. 8.

Fig. 7.—Same case as Fig. 6. Shows loss of surface keratin, and hydropic alteration of the stratum spinosum, hyperplasia of the basal cell layer, and inflammatory changes in the lamina propria. (Magnification ×294.)

Fig. 8.—High power of Fig. 7. Shows pigmentation in cytoplasm of basal cells which began during pregnancy but did not regress following parturition. This pigmentation produces the black color seen in the proliferative tissue clinically. (Cloasma gravidarum.) (Magnification ×2360.)

Class V: The fifth form of pregnancy gingivitis is the so-called pregnancy tumor, for the most part confined to a single growth springing up at any point in the mouth. At the outset, it appears to be an overgrown gum papilla. After it has attained considerable size, it resembles an epulis varying from 1 to 2 cm. in diameter, and sometimes larger. It usually has a pedunculated attachment. In color, it is cyanotic, with a bright red border. Often it interferes with mastication, and the trauma to which it is subjected produces an area of grayish necrosis on parts of the surface. However, in one case studied, the entire growth was blood red. Here also, gingival irritants may be present. The tumor may begin early in pregnancy, and usually grows rapidly and easily. It differs

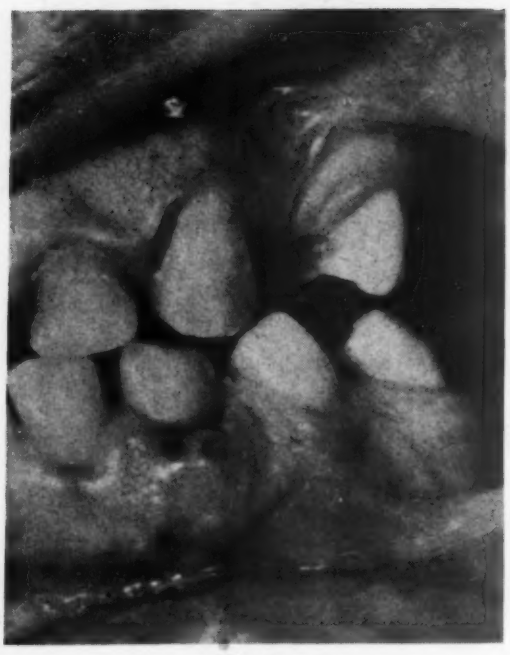


Fig. 9.

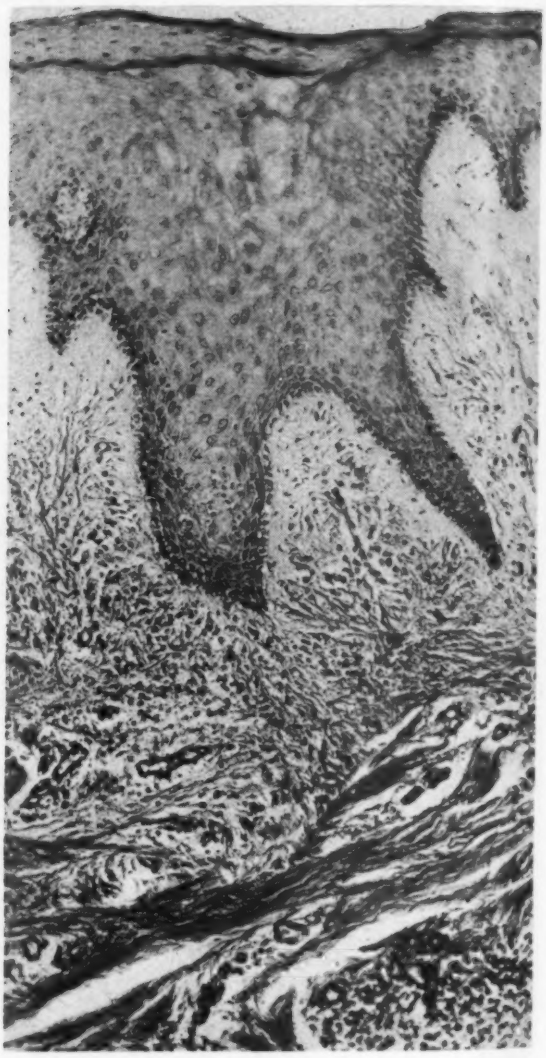


Fig. 10.

Fig. 9.—H. G., aged 28 years, Negro woman, multipara, eighth month. Class IVD. Shows proliferation starting from the undersurface of the free gum margin around the lower left deciduous cuspid, but not involving the interdental papilla.

Fig. 10.—Same case as Fig. 9. Shows parakeratosis of the epithelium, hyperplasia of basal cell layer, and inflammatory changes in the lamina propria. (Magnification ×168.)

Fig. 11.

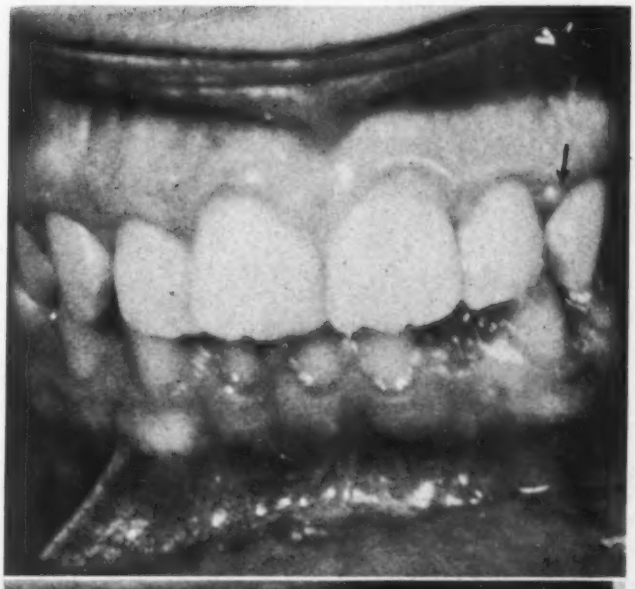




Fig. 12.

F.g. 11.—D. H., aged 17 years, white, primipara, sixth month. Class IVE. Shows the formation of a pseudopapilla between the upper left lateral incisor and cuspid. The interdental papilla is not enlarged and the pseudopapilla is of the same color as the rest of the gingiva, but a definite line of separation exists.

Fig. 12.—A. G., aged 25 years, white, sixth month. Class V. Shows a so-called "pregnancy tumor" between the lower central incisors. This type of pregnancy tumor is not confined to any location.

from the ordinary epulis in that it may either disappear entirely or diminish greatly in size after parturition. (Figs. 12, 13, and 14.)

While the pregnancy tumor is identified with the pregnant state, a true epulis may be coexistent. The epulis may likewise grow with pregnancy and diminish with parturition, but it has other distinguishing characteristics and should not be confused with the condition known as pregnancy tumor. (Figs. 15, 16, 17, and 18.)

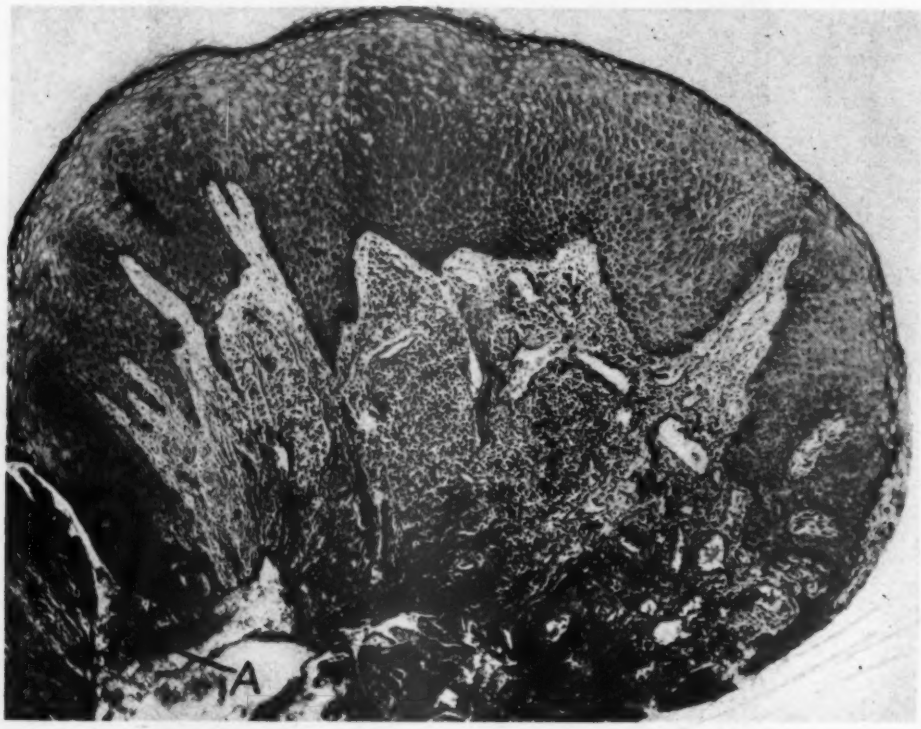


Fig. 13.—Same case as Fig. 12. Shows loss of the surface keratin, extensive acanthosis and hydropic change of the stratum spinosum, and inflammatory exudate and increased vascularity in the lamina propria. The line of demarcation between normal and pathologic tissue can be noted at *A*.

B. *Microscopic Changes During Pregnancy.*—In the epidermis, the most striking change was the loss of surface keratin (Fig. 19), which became more marked as pregnancy advanced from the fourth to the ninth month. However, in most cases slight keratinization reappeared in the ninth month. In some cases, a zone of parakeratosis was seen, varying in depth up to four or five cells (Fig. 10). In two instances, a keratin layer was seen on the surface of the areolar gingivae where none is seen normally (Fig. 38). In general, the areolar gingivae (mucous membranes) showed the other changes similar to the alveolar gingivae, but not to the same degree (Fig. 21). In the stratum spinosum there was extensive hydropic change with pyknotic degeneration of the nuclei toward the surface (Fig. 20). There was an acanthosis present, as well as hyperplasia of the stratum germinativum (Fig. 19). The peg patterns were elongated, penetrated deeply into the lamina propria, and were pointed and split frequently (Fig. 13). No increased mitosis was seen. Pearl formation was seen in four of the cases.

Fig. 14.

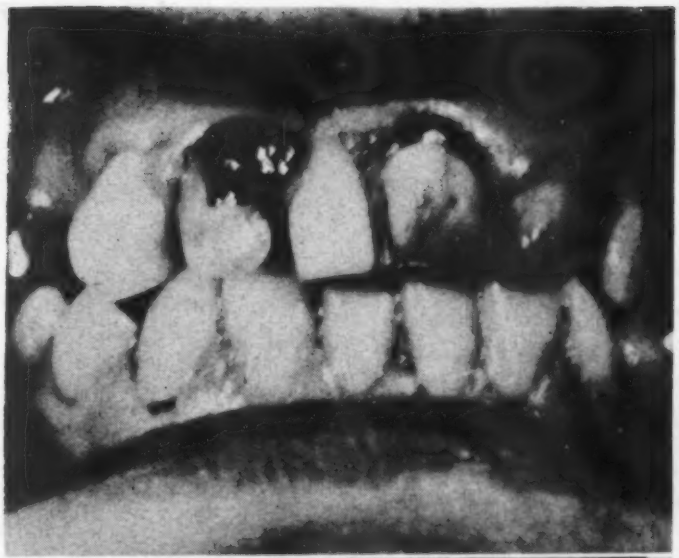




Fig. 15.

Fig. 14.—Y. G., white, multipara, seventh month. Class V. Shows a so-called "pregnancy tumor" between the upper right central and lateral incisors.

Fig. 15.—E. F., white, age 35, multipara, fifth month. Coexistent epulis. Soft oblong mass, located between the lower right lateral incisor and cuspid. It was about 1.5 cm. long in vertical diameter, and 0.75 cm. in horizontal diameter. It was bright raspberry red in color, the entire surface appearing smooth with a very fine gray network appearing on the surface on the lower two-thirds, giving that part a reticulated appearance. It was movable and free from the gum except at the middle where it appeared to be attached to the gingival margin by pedicle. It was not painful. The lesion bled easily upon traumatization. It first appeared in this location about two years previously. The growth was gradual and did not then attain its present size. It was removed and diagnosed as an epulis or fibroid tumor. Following its removal, there was no regrowth for about six months, after which time it recurred. There was a second removal about one year ago. Following this operation, there was freedom from the growth for about ten months; about one month previously, the epulis returned and was enlarging at a greater rate than heretofore. The patient was in the fifth month of her third pregnancy. Previous pregnancies occurred nine and five years ago. The patient did not have any similar lesions during the previous pregnancies.

In the lamina propria, the blood vessels were dilated (Fig. 13). The amount of collagen and connective tissue varied in the different cases, so that no conclusion could be drawn as to the effect of pregnancy on these structures. There

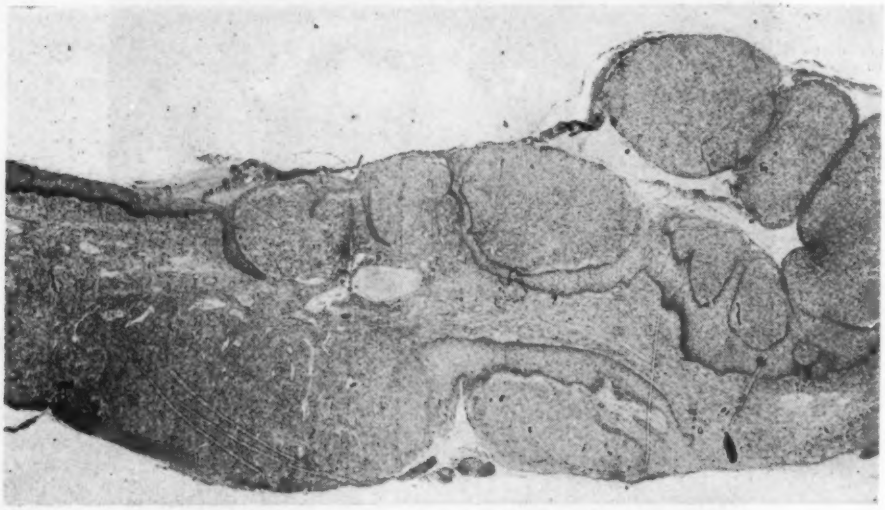


Fig. 16.—Same case as Fig. 15. Photomicrograph shows growth on top of mucous membrane. (Magnification ×28.)

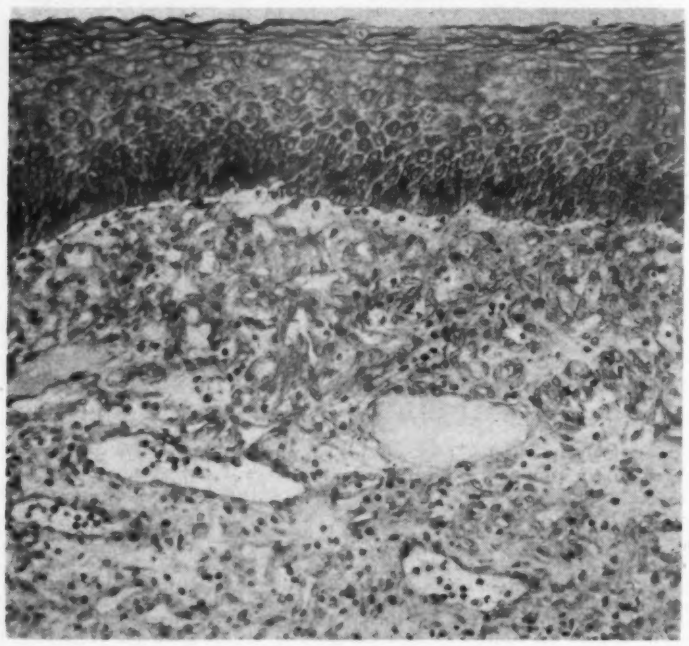


Fig. 17.—High power of Fig. 16. Shows mucous membrane, to which tumor was attached. The changes were characteristic of pregnancy gingivitis. (Magnification ×240.)

was heavy cellular inflammatory infiltration which included plasma cells, lymphocytes, leucocytes, and occasional eosinophiles (Figs. 13, 19, and 20). In most instances, the perivascular infiltration was seen chiefly near the crest of the

interdental papilla, but in some cases it was distant from the areas commonly recognized as subject to irritation (Figs. 22 and 23).

There were evidences of edema in both the epithelium and the lamina propria. The epithelium stained relatively poorly. In general, the tissue was a degenerative one with keratin loss, edema and hydropic alteration and pyknosis, but there was also hyperplasia present. The "sprouting" granulation tissue seen clinically in Figs. 4 and 5 is characterized microscopically by

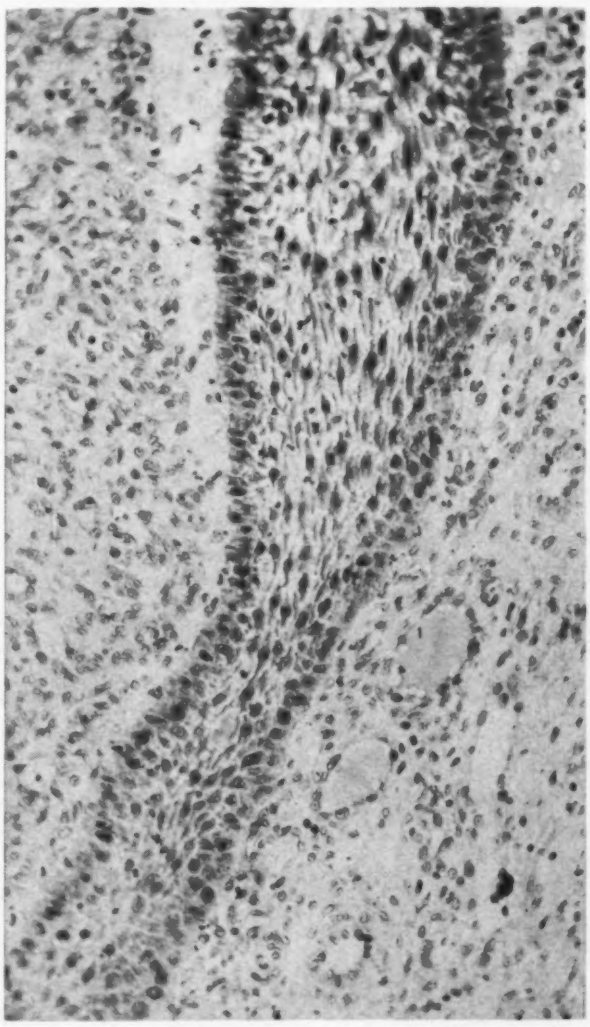


Fig. 18.—Higher power of Fig. 16. Shows marked increase in vascularity, edema and hydropic degeneration, cellular infiltration and acanthosis, hyperplasia of basal cell layer.

a proliferative tissue composed of inflammatory cells, fibroblasts with newly formed collagen, and newly formed capillaries (Fig. 19 at *A*). The formation of the pseudopapillae seen clinically in Figs. 2 and 6 is characterized by the outgrowth of the tissues as in Fig. 24, 25, and 26. An unusual type of degeneration characterized by the formation of hyaline bodies in the epithelium near the surface was seen in one case (Fig. 27). The microscopic gingival changes in the twenty pregnant women are listed in Table I.

DISCUSSION OF ENDOCRINE RELATIONSHIPS

A. *Hypophysis.*—Experimental and clinical work indicates that the anterior lobe of the pituitary gland influences or controls the other glands of internal secretion (Smith, 1942). Hence a consideration of this gland forms a valuable starting point in determining endocrine relationships. The activity of the hypophysis is demonstrated by the striking changes resulting from extensive functional deficiency of this gland in man (Simmond's disease) and from hypophysectomy in animals. In Simmond's disease and following hypophysectomy, there is an involution of the thyroid gland, the adrenal cortex, the gonads, and the accessory sex organs; the capacity for muscular work is decreased; circulation is sluggish; blood pressure and basal metabolism are lowered; and resistance to infection and trauma is diminished (Smith, 1942).

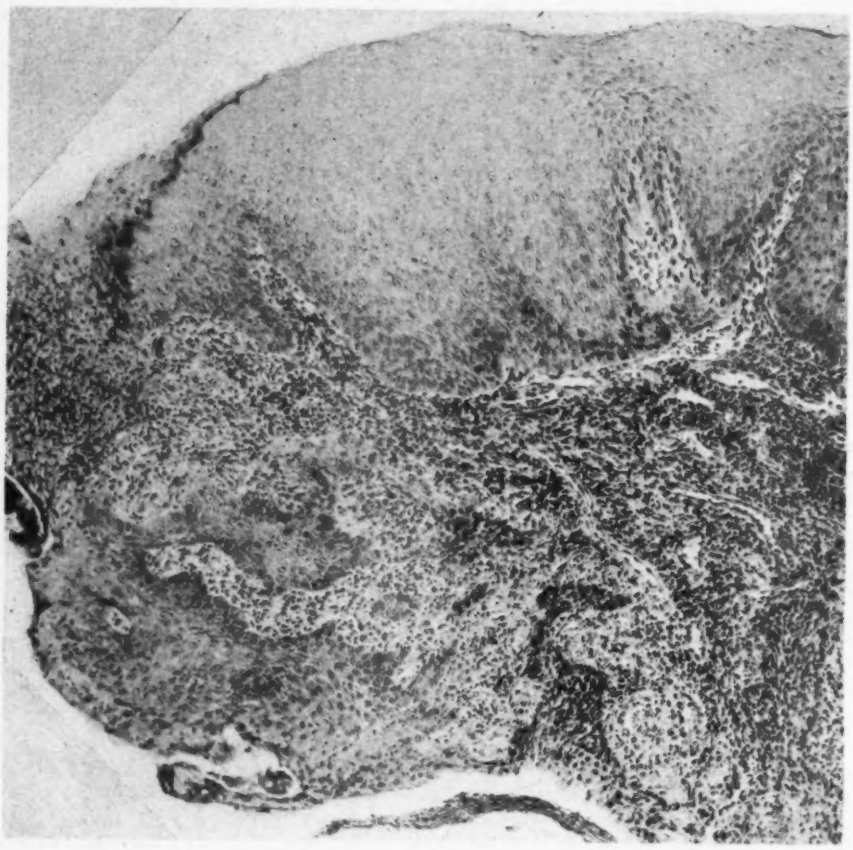


Fig. 19.—B. E., aged 27 years, white, eighth month, multipara, shows the proliferative changes in the alveolar gingiva at the tip of the papilla as seen in Class IVB. Marked loss of surface keratin, hyperplasia, and inflammatory exudate in lamina propria. (Magnification ×138.)

The thyroid hormone has a depressant action on the thyrotropic function of the hypophysis, since, in hyperthyroidism, subnormal quantities of thyrotropic substances are found in the urine (Emerson and Cutting, 1938; Uotila, 1940). Conversely, hypophysectomy, or Simmond's disease, causes an involution in the thyroid; myxedema may be the dominant feature (Smith, 1930; Means, Hertz

and Lerman, 1940). Thyroidectomy causes a decrease in the number of acidophiles and an apparent increase in the chromophobes, probably due to degranulation of the acidophiles, an indication of decreased activity. These changes may be prevented or reversed by thyroxin therapy (Marine, Rosen, and Spark, 1935).

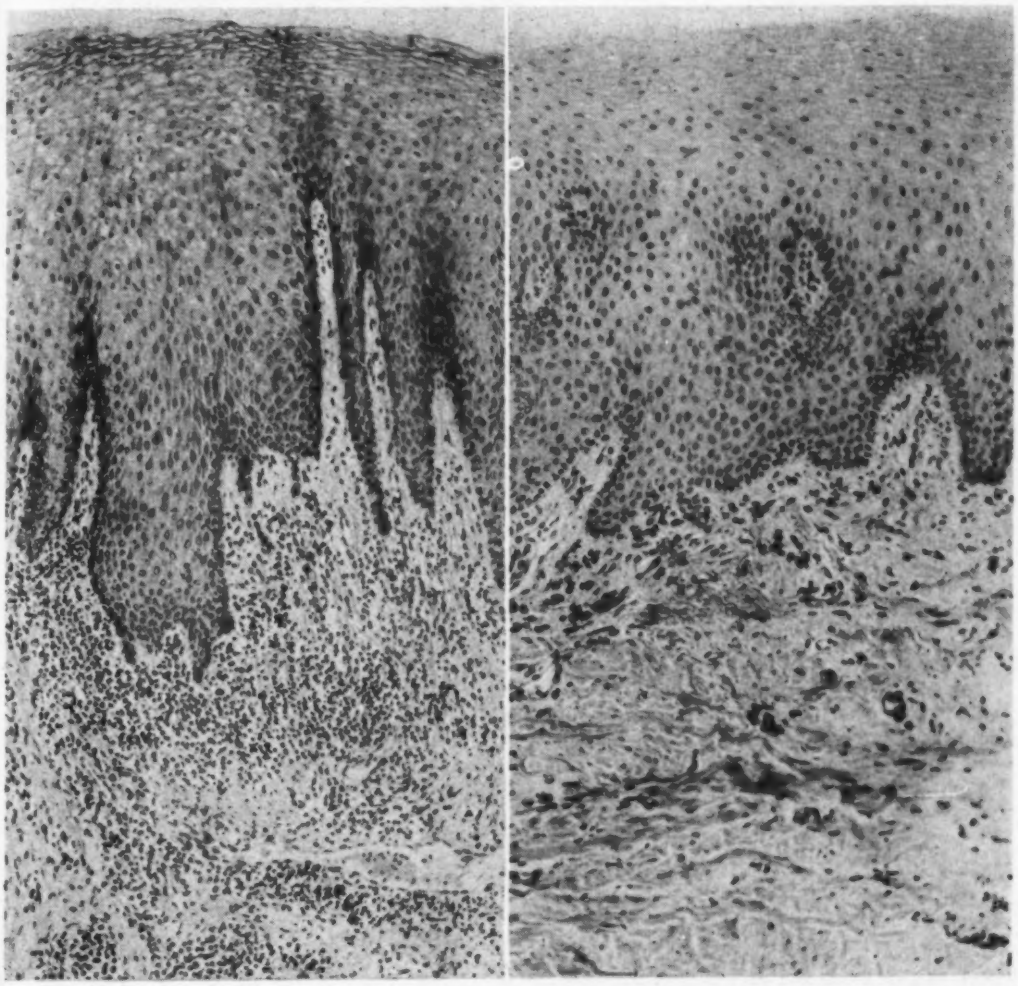


Fig. 20. Fig. 21.

Fig. 20.—Same case as Fig. 19. Shows the alveolar gingiva of upper right cuspid with characteristic degenerative changes, namely, loss of keratin, extensive hydropic degeneration, and pyknosis. There is an extension of epithelial pegs into the dermis and extensive inflammatory infiltration. (Magnification ×150.)

Fig. 21.—Same case as Fig. 19. Shows the mucous membrane. The hydropic degeneration and inflammatory changes in the lamina propria are not as marked as in the alveolar gingiva but are present. (Magnification ×189.)

There is also a reciprocal relationship between the adrenal cortex and the hypophysis. Hypophysectomy causes a decrease in the size of the adrenals (Smith, 1930). Conversely, acidophile adenomas produce an excess of adrenotropic factor since adrenal hypertrophy usually occurs in acromegaly (Goldzieher, 1944). Adrenalectomy causes a decrease in the number of basophile

cells of the hypophysis (Crooke and Russell, 1935), although overactivity of the adrenal gland (Cushing's syndrome) also causes basophile degeneration of the hypophysis, owing either to overactivity of the basophiles, or a specific degenerative effect of abnormal cortex secretion (McLetchie, 1942; Goldzieher, 1944).

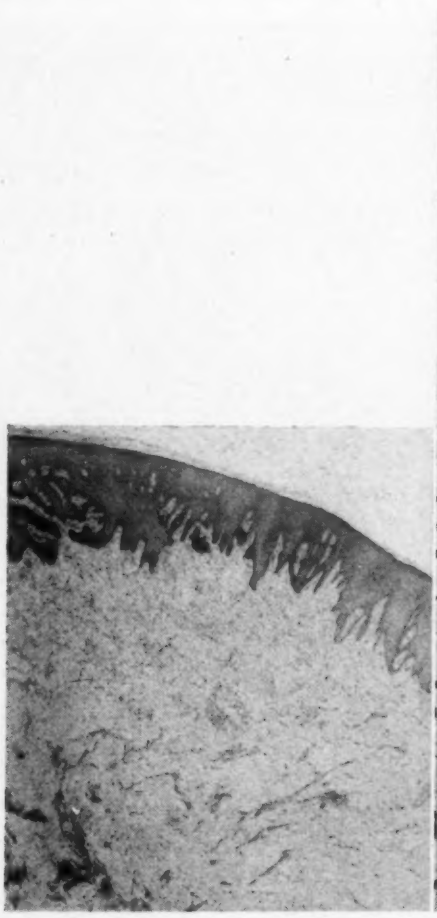


Fig. 22.

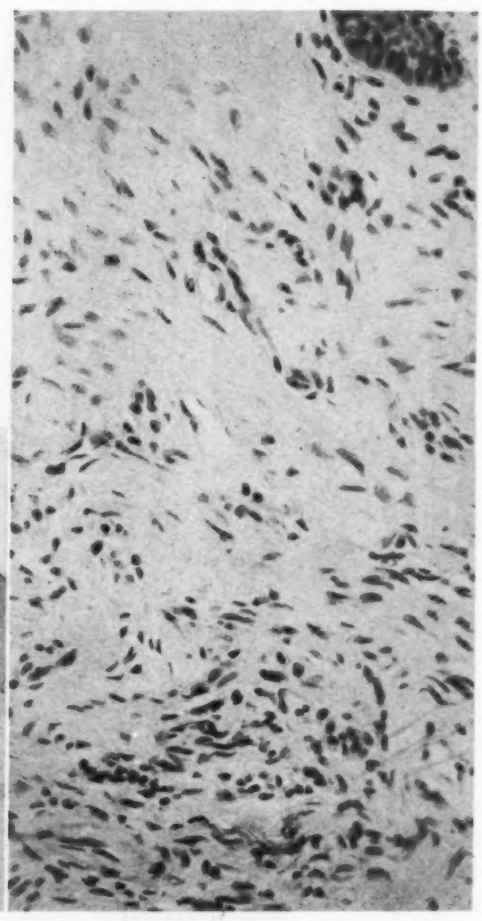


Fig. 23.

Fig. 22.—Same case as Fig. 15. Shows section of alveolar gingiva in an edentulous area. There is parakeratosis and acanthosis present and hyperplasia of the basal cell layer. Foci of inflammation are seen in lamina propria. (Magnification ×50.)

Fig. 23.—High power of Fig. 22. Shows connective tissue with increase in capillaries, inflammatory exudate, and hyperemia. (Magnification ×376.)

Hypophyseal factors are necessary for maintenance of the gonads, and through them, of the accessory sex organs. Hypophysectomy causes atrophy of the gonads, and, conversely, atrophic glands of hypophysectomized rats can be repaired by injection of gonadotropic substance (Smith, 1939). It is generally believed that two gonadotropic principles—a follicle-stimulating factor and a luteinizing factor—are elaborated by the hypophysis (Greep, van Dyke, and Chow, 1942), but Smith (1942) believes that the determination of the number of gonadotropic hormones has not as yet been made.

During pregnancy, there is an increase in size and weight of the hypophysis. Cytologically, there is an active secretion, as indicated by granules, hypertrophied Golgi apparatus, and abundant mitochondria (Rasmussen, 1933). The interpretation of the cytological changes observed during pregnancy is conflicting. However, Severinghaus (1938, 1939) states that in pregnancy both the acidophiles and basophiles are actively secreting, and, therefore, the so-called pregnancy cell is not a distinct cell type but rather some phase of the active acidophile or basophile secretory cycle. Although the hypophysis is considered to be in a hyperactive state during pregnancy, it has been shown that between the second month of pregnancy, and the week following parturition, the gland has little or no gonadotropic potency (Saxton and Loeb, 1937; Witschi and Riley, 1940). The increased cellular activity, together with the decreased gonad-stimulating potency of the gland tissue itself, indicates a rapid release of secretory products.

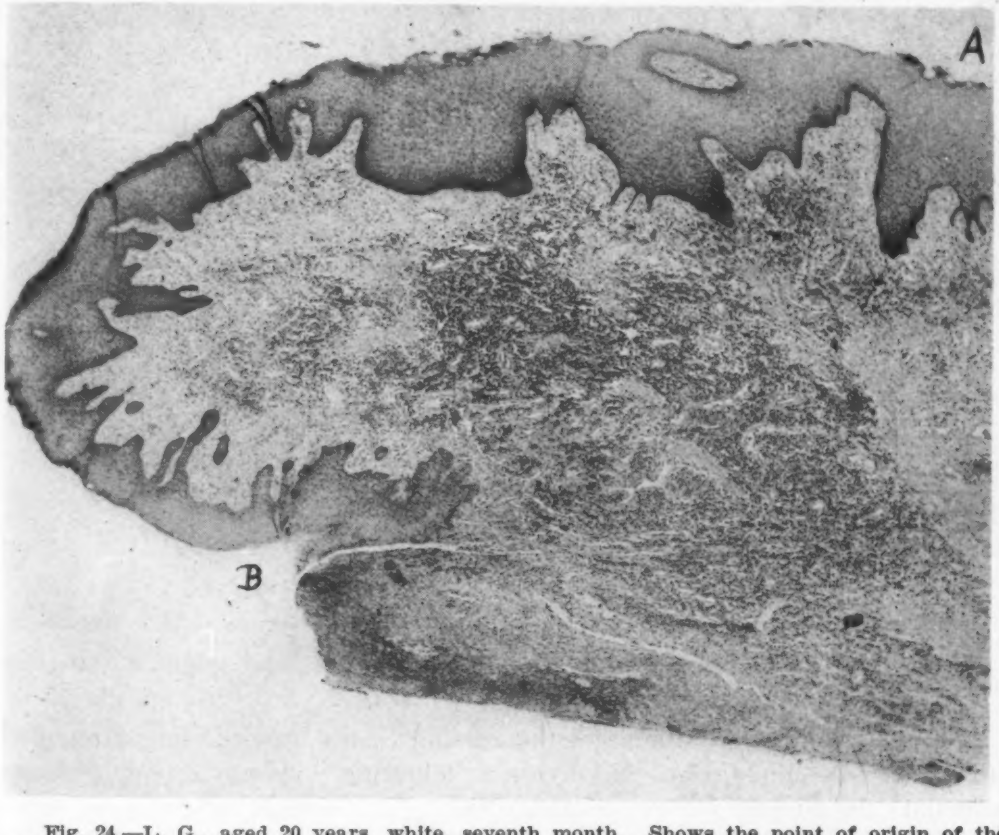


Fig. 24.—L. G., aged 20 years, white, seventh month. Shows the point of origin of the proliferation of a pseudopapilla forming at the gingival crevice and forcing the normal gum back at *A*. A second outgrowth is seen at *B*. (Magnification ×47.)

Following hypophysectomy, there are extensive degenerative changes in the gingivae and oral mucous membranes of female rhesus monkeys. The keratin layer is for the most part absent, the prickle cell layer shows marked evidence of degeneration, the cells appear irregular in shape and size, and the intercellular bridges are poorly preserved (Ziskin and Blackberg, 1940).

B. *The Thyroid Gland.*—Closely associated with pituitary function is the activity of the thyroid gland (Colly, 1942). The thyroid gland has manifold effects on diverse organs and functions. It acts on growth, maturation, and differentiation of tissue; on the distribution of water, salts, and colloids of the body; on carbohydrate metabolism and on the nervous, muscular, and circulatory systems (Lerman, 1942).



Fig. 25.



Fig. 26.

Fig. 25.—High power of Fig. 24. Shows point of first outgrowth at *A*. (Magnification ×86.)

Fig. 26.—Higher power of Fig. 24. Shows point of second outgrowth at *B*. (Magnification ×120.)

The thyroid gland stimulates the adrenal cortex by mediation through the pituitary. Administration of thyroxin following hypophysectomy does not stimulate the adrenal cortex (Swann, 1940). The cortical hypertrophy elicited by thyroxin is an expression of increased hormone production needed to compensate for the effects of thyroxin (Goldzieher, 1944).

Although much clinical data closely correlates thyroid and gonadal functions, no specific hormonal connections have been verified between these glands (Hamblen, 1945). However, Tyndale and Levin (1937) have found that thyroxin decreases the ovarian response of hypophysectomized rats to injections of extracts of urine of castrated women and concluded that thyroxin inhibits

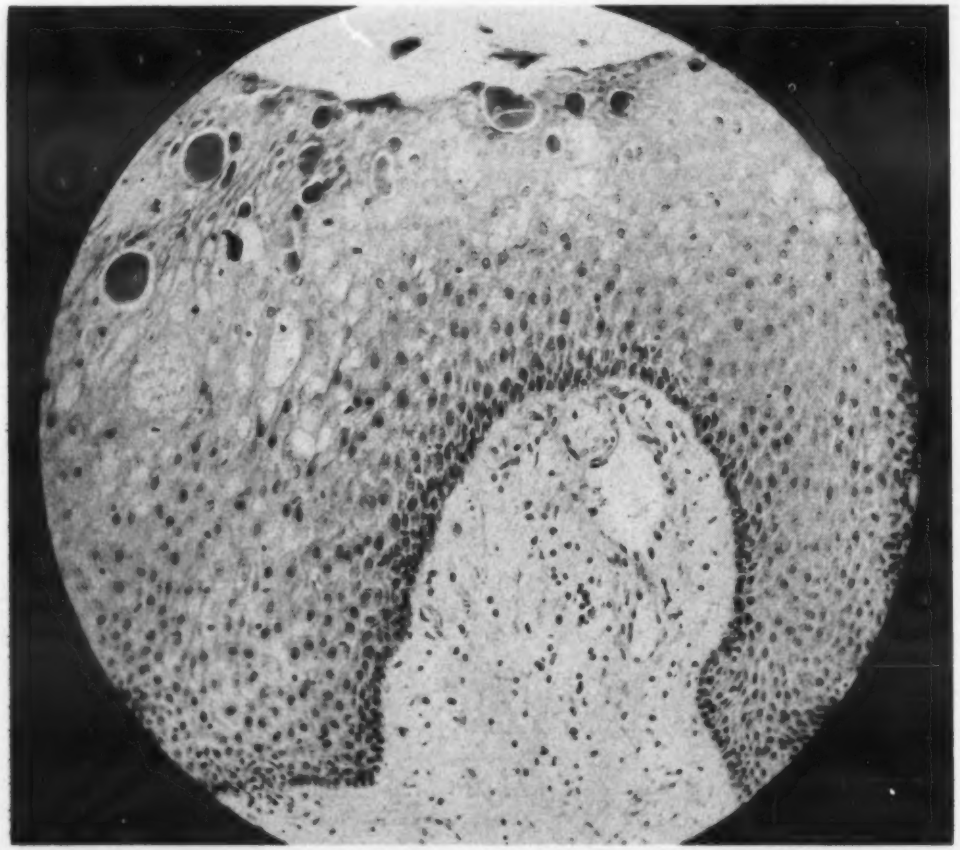


Fig. 27.

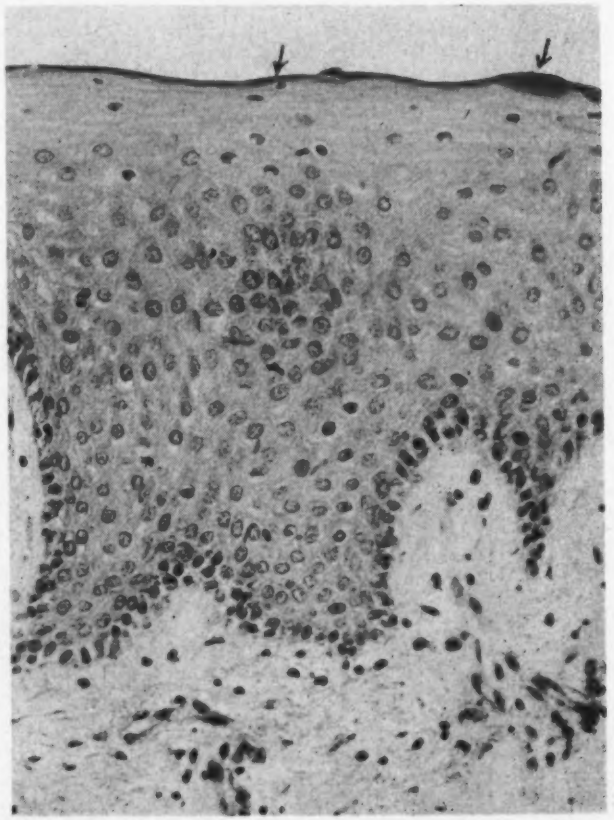


Fig. 28.

Fig. 27.—Same case as Fig. 24. Shows degenerative changes and hyaline bodies near middle of alveolar gingiva. Compare with hyaline bodies seen in case of thyroid dysfunction in Fig. 41. (Magnification ×224.)

Fig. 28.—Same case as Fig. 24. Shows keratin and keratin curl on areolar gingiva where keratin is not found normally. (Magnification ×390.)

the gonads directly. In any case, the action of thyroxin may be such as to produce a state of low estrogenic function (Lerman, 1942).

During pregnancy, there is an enlargement of the thyroid gland caused by parenchymatous changes, increased vascularity, and epithelial hyperplasia (Hughes, 1940). There is an increase in the basal metabolic rate because of increased demands on the part of the fetus (Plass and Yoaken, 1929; McLaughlin and McGoogan, 1943).

Thyroidectomy in rhesus monkeys causes hyperkeratosis in the alveolar and areolar gingivae, epithelial intercellular edema, increased mitosis of the basal cell layer, and deposition of mucous-staining material and glycogen. Inadequately treated cretins and myxedematous humans show similar changes (Ziskin and Stein, 1942).

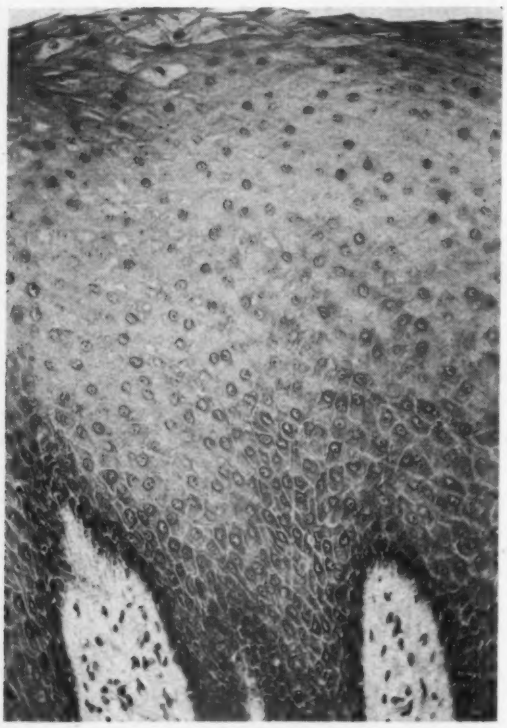


Fig. 29.—A. T., case of hirsutism and unknown glandular dysfunction. Section of the alveolar gingiva of upper right lateral incisor showing epithelial degeneration consisting of keratin reduction and hydropic alteration. (Magnification ×224.)

C. *The Adrenal Glands.*—Among the known physiologic activities affected by the adrenal cortex are carbohydrate metabolism, capacity of muscles to respond to stimulation, distribution of electrolytes, and resistance to stress. The interrelationship between the adrenals and the other glands of internal secretion is reciprocal, and often indirect, with the hypophysis acting as the mediator (Goldzieher, 1944).

The gonads are closely associated with the adrenal cortex; estrone, progesterone, and various androgens have been found in extracts from normal adrenal glands. Organic or functional changes in the adrenal cortex which

produce abnormal changes in the sexual sphere are referred to as the adrenogenital syndrome. However, since the changes vary with the type of hormone produced, the age and the sex of the patient, several syndromes can be distinguished (Goldzieher, 1944).

During pregnancy, the adrenals are enlarged, but the diffuse hyperplasia is functional and disappears after parturition; the change is a physiologic one, its mechanism being uncertain.

In Addison's disease, there is increased thickness of the surface keratin of the alveolar gingivae, parakeratosis, hyperplasia of the basal cell layer, and a great increase of connective tissue (Figs. 42 and 43). In adrenal hyperplasia, however, there is a reduction of the surface keratin, pyknosis and hydropic degeneration in the stratum spinosum, and hyperplasia of the basal cell layer (Figs. 44 and 45).

Fig. 30.



Fig. 31.

Fig. 30.—Monkey 144, pre-experimental, before treatment with estrogen. Note inflamed condition of gingiva and compare with Fig. 31.

Fig. 31.—Monkey 144, postexperimental, following twenty-five days of treatment with estrogen. Note reduction of inflammatory changes around the gingivae and thickening of epithelium.

D. *The Ovaries.*—In the childbearing age, the ovaries produce two principal hormones, estrogen and progesterone. Estrogen, secreted by the developing follicle, is essential for the growth and development of the accessory sex organs in the female. Progesterone, secreted by the corpus luteum, varies in its relation to estrogen, sometimes acting synergistically and sometimes antagonistically (Allen, 1939).

Under physiologic conditions, the circulating estrogens are destroyed in the liver by oxidation (Zondek, 1934; Israel, Meranze, and Johnston, 1937; Segaloff, 1943). Pregnancy, per se, does not alter the liver metabolism of estrogens in vitro, since liver sections of pregnant rats metabolize estrogens to the same extent as liver sections of nonpregnant rats (Heller and Heller, 1943). However, Cantarow and co-workers (1942, 1943) have set forth the hypothesis that an enterohepatic circulation occurs with estrogens similar to that of the bile acids.

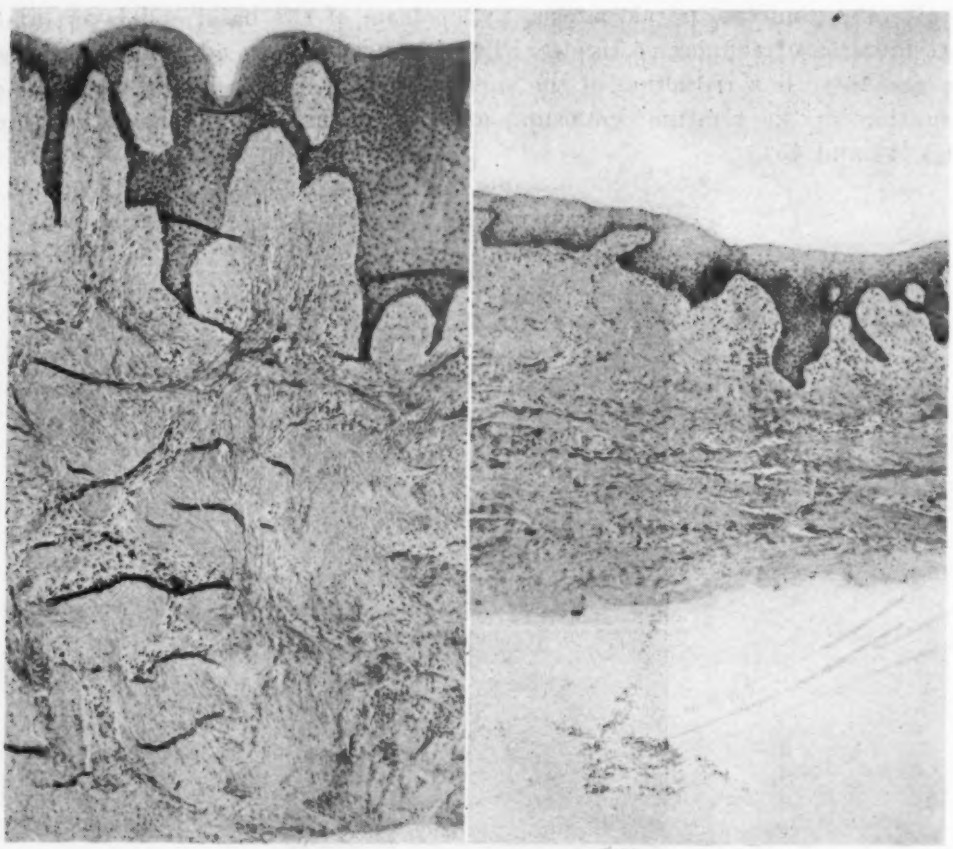


Fig. 32. Fig. 33.

Fig. 32.—B. H., aged 41 years, treated with diethylstilbesterol for two years for a gynecologic disorder. Shows great increase in thickness of lamina propria and downgrowth of epithelium in alveolar gingivae. (Magnification ×69.)

Fig. 33.—Same case as Fig. 32. Shows analogous changes in the areolar gingiva, namely, hyperplasia of the epithelium and lamina propria. (Magnification ×69.)

Normally, only relatively small amounts of estrogen and progesterone are found in the urine. During pregnancy, however, there is a marked increase in the blood level and urinary excretion of both estrogen and progesterone. The estrogen content of pregnancy urine rises gradually until parturition, and then decreases markedly in the course of a few days. There are three forms of estrogen; in order of decreasing physiologic activity these are: estradiol, estrone, and estriol. In the latter months of pregnancy, the least potent form, estriol, is present in the greatest amount. At term, however, it disappears from the

urine and estradiol rises suddenly in concentration (Smith and Smith, 1941). Also, during the first eight months of pregnancy, 99 per cent of the estrogen is excreted conjugated with glucuronic acid as the glucuronide, which is an inactive detoxified form, but shortly before parturition the amounts of unconjugated active estrogen increase markedly (Cohen, Marrian, and Watson, 1935). In addition, the estrogens of pregnancy serum appear to be intimately bound to the protein fraction, as indicated by the facts: (1) that they do not pass through a collodion membrane, (2) that they are precipitated in the protein fraction,

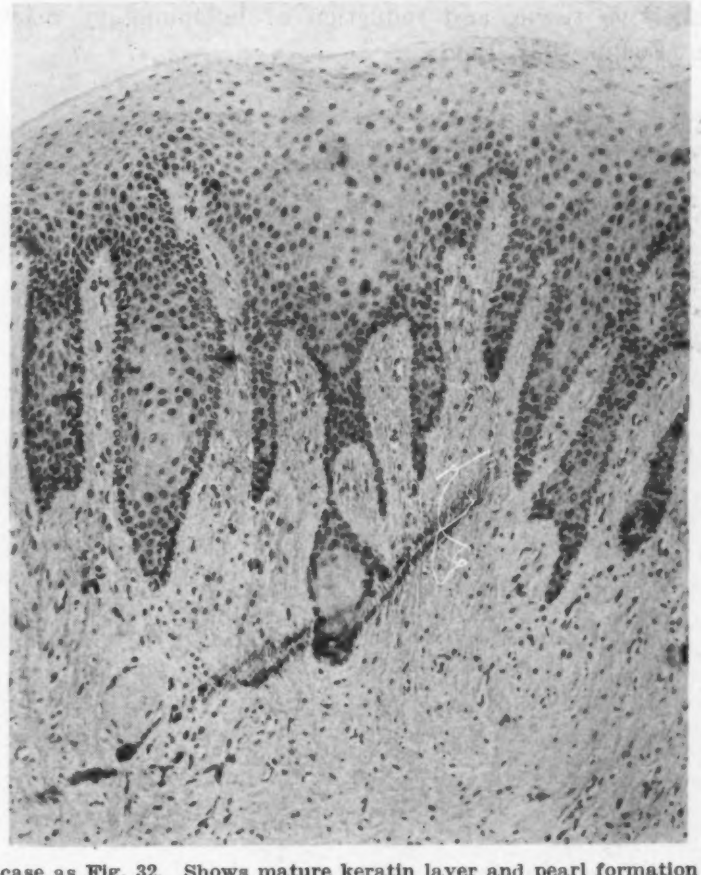


Fig. 34.—Same case as Fig. 32. Shows mature keratin layer and pearl formation in the alveolar gingiva. (Magnification ×175.)

and (3) that prolonged hydrolysis is necessary to liberate them from the protein fraction. The amount of protein-combined hormone ranges from 23 to 50 per cent of the total estrogens (Mühlbock, 1939; Rakoff, 1943). This evidence indicates that although estrogen is found in increased amounts in the urine and serum during pregnancy, most of it is present in a bound inactive form. Despite these factors, the total secretion of estrogen is so great that sizable quantities of free estrogen are available for body needs.

Progesterone does not increase in amount in the urine during the first sixty days of gestation, but it then rises until a peak is reached in the eighth month (Browne, Henry, and Venning, 1937). The conversion of estrone to

estriol is facilitated by the action of progesterone, which also acts to prevent the catabolism of estrogen compounds; an imbalance of estrogens may occur during pregnancy as a result of progesterone deficiency (Smith and Smith, 1941).

Castration, in female rhesus monkeys, causes alteration of the arrangement of the prickle cell layer of the alveolar and areolar gingivae, and tissue degeneration (Ziskin and Blackberg, 1940). The injection of estrogenic hormones, however, has a beneficial effect on the gingivae and oral mucous membranes. Estrogenic injection produces increased keratinization, hyperplasia of the prickle cell layer and connective tissue, and reduction of inflammatory reactions in the lamina propria (Ziskin, 1937, 1938).

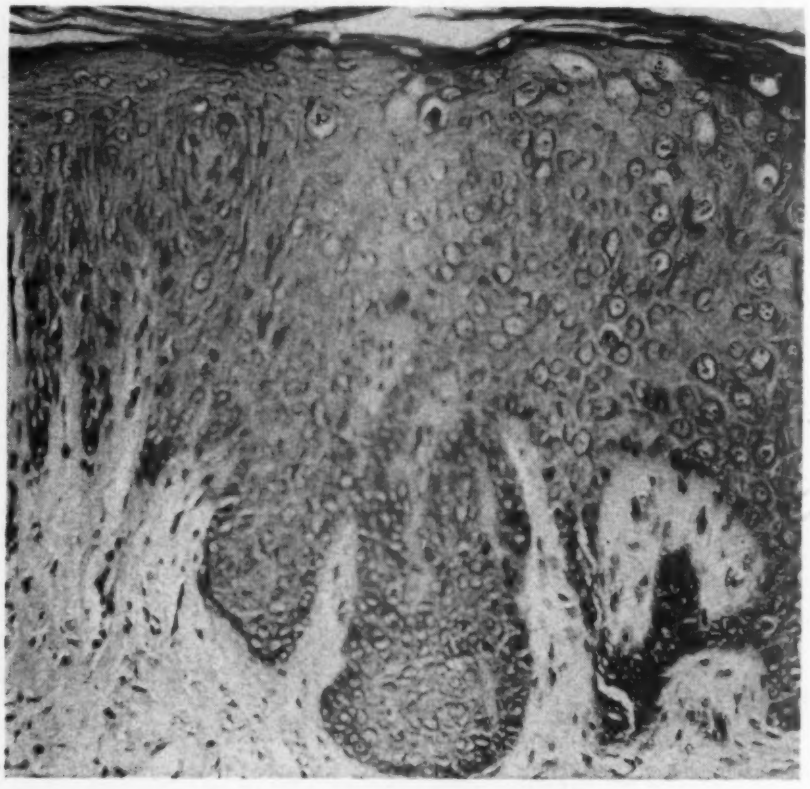


Fig. 35.—Monkey 123, pre-experimental, before treatment with chorionic gonadotropin. Note the normal condition of the keratin and the rest of the epithelium. (Magnification ×300.)

E. *The Placenta.*—Another organ of unquestionable importance during pregnancy is the placenta, although the proof of its specific relations is as yet obscure. The placenta is a source of extraovarian estrogen found in pregnancy. The high value of urinary estrogen which rapidly drops to normal following parturition, the continued presence of estrogen in pregnancy following oöphorectomy, and the high concentration of estrogen in the placenta itself (Allen, Pratt, and Doisy, 1925), all indicate that the placenta is a source of estrogen. The placenta is probably also concerned with the production of progesterone. Progesterone is found in pregnancy after removal of the ovaries, and placental

extracts have luteal activity, thus suggesting the placenta as an extraovarian source of this hormone (McGinty, McCullough, and Wolter, 1936).

Another substance present in the urine during pregnancy and of no known metabolic function is the hormone known as chorionic gonadotropin. It has an action on the ovary of rodents (which forms the basis of the various pregnancy tests), but this action differs from that of the gonadotropic principle of the hypophysis (Engle, 1933). The secretion of chorionic gonadotropin seems to be very clearly related to the placenta, since the hormone is found in the urine of men having a chorioepithelioma and is definitely associated with chorionic tissue (Ware and Main, 1934). It has been shown by Browne and Venning (1936) and Evans, Kohls, and Wonder (1937) that the chorionic gonadotropin excretion rises spectacularly to a rapid peak which is reached between the twentieth and fiftieth day of pregnancy. The chorionic gonadotropin then declines rapidly to relatively low levels which are maintained until a few days after parturition.



Fig. 36.—Monkey 123, postexperimental, following sixty-eight days of chorionic gonadotropin injections. Note the loss of mature keratin, a parakeratosis, and the epithelial hyperplasia without inflammatory cells. (Magnification ×160.)

The effect of the administration of chorionic gonadotropin in women differs markedly from that of the gonadotropic principle of the anterior lobe of the pituitary gland. Geist (1933) has reported that the administration of chorionic gonadotropin causes follicular damage and no stimulation, but rather an arrest

of follicular development. Hamblen (1935) reports that chorionic gonadotropin produces no stimulation of the early follicle and increases degenerative and cystic changes in the mature ones.

Chorionic gonadotropin administration causes gross and microscopic degenerative changes in the gingivae and oral mucous membranes of women patients and rhesus monkeys. Grossly, there is edema, inflammation, and increased tendency to bleeding, which takes on the appearance of Vincent's infection. Microscopically, there is reduction of surface keratin, cellular deterioration of the stratum spinosum, and subepithelial inflammation (Ziskin, 1937, 1938).

F. *Vitamins.*—Of increasing significance is the realization of a correlation between the hormones and the vitamins, since in the diet which is inadequate for proper nutrition, there may be endocrine imbalance. A discussion of the many effects of vitamin deficiency is beyond the scope of this paper, but the relationships which have been shown between some of the vitamins and the endocrine system will be reviewed.

Vitamin A deficiency causes a characteristic benign metaplasia, keratinization, and loss of secretory function in the epithelial tissues throughout the body. The vitamin probably acts as a catalyst or regulator of certain metabolic processes peculiarly essential to the functional and structural integrity of epithelial cells. The source of vitamin A in the body appears to be a pigment, carotene, which is converted by enzyme action to vitamin A, the vitamin itself being stored in the liver. Wohl and Feldman (1939) have shown that the thyroid hormone is necessary for carotene conversion, and also for the storage of vitamin A in the liver. Some investigations have reported an antagonism between vitamin A and thyroxin, but the evidence is not as yet conclusive. Ziskin, Rosenstein, and Drucker (1943) have found that vitamin A suppresses or ameliorates the keratinization and hyperplasia produced by estrogen injections, thus indicating an inverse relationship between estrogen and vitamin A, and suggesting that estrogen may induce keratinization and hyperplasia in epithelial structures in a manner resembling vitamin A deficiency.

During pregnancy, there is a decrease in the plasma level of vitamin A in the third trimester. Storage in the fetal liver and utilization by the fetal tissues are suggested as causes of the low vitamin plasma level (Bodansky, Lewis, and Lillienfeld, 1943). This may or may not have a bearing on the increase in gingival keratinization during the last month of the term.

There is a variety of diseases caused by deficiency of the members of the vitamin B complex, the most important of which are beriberi (thiamin deficiency), cheilosis (riboflavin deficiency), and pellagra (niacin deficiency). Experimental work indicates an inverse relationship between the vitamin B complex and the thyroid gland. Vitamin B ameliorates the weight loss induced by hyperthyroidism (Himwich, 1932); the kidneys and livers of hyperthyroid animals are depleted of vitamin B (Drill, 1938); abnormal liver function is produced by thyroid feedings when the yeast content of the diet is inadequate (Drill and Hayes, 1940).

Within the past few years a relationship between vitamin B complex and the estrogens has been established. Sutton and Ashworth (1940) have shown that acute glossitis and pellagra lesions were ameliorated by an extract of the anterior lobe of the pituitary gland after failure to respond to nicotinic acid, riboflavin, parenteral liver, and adequate diet, thus indicating a relationship between the vitamin B complex and the anterior lobe of the pituitary gland. The same authors (1942) reported that administration of estrogens to persons with subclinical vitamin B complex deficiency may cause the appearance of lesions characteristic of thiamin deficiency, niacin deficiency, and riboflavin deficiency. They suggest that the anterior lobe of the pituitary secretes a hormone which facilitates utilization of the vitamin B complex, and that estrogen either increases the demand for this hormone or suppresses its secretion.



Fig. 37.—Monkey 118 treated with chorionic gonadotropin for sixteen days and photographed eight days following end of treatment. Shows proliferation tissue formed around upper left central incisor. Inflammatory changes around upper anterior teeth. Clinically the picture was that of subacute Vincent's infection, whereas before treatment the gingivae were essentially normal.

Singher et al. (1944) and Segaloff and Segaloff (1944) have shown that deficiency in the vitamins of the B complex—principally thiamin and riboflavin—decreases the ability of the rat liver to inactivate estrogens both in vivo and in vitro. Biskind and Shelesnyak (1942) and Biskind (1942) have shown that the liver has an important function in maintaining the estrogens in the body at a normal level, and that avitaminosis B impairs liver function, thus causing an excess of estrogen in the body. They have found that patients with vitamin B deficiency may have certain clinical conditions which can be caused by an excess of free estrogens, such as menorrhagia, metrorrhagia, cystic mastitis, and premenstrual tension, and that these conditions are improved by the administration of adequate doses of the vitamin B complex.

During pregnancy, there is increased need of thiamin, lack of which may cause a thiamin avitaminosis with evidences of neuritis (Schute, 1942, and Lockhart, Kirkwood, and Harris, 1943). Under conditions of diet in which thiamin

Fig. 38.



Fig. 39.



Fig. 38.—D. S., white, aged 15 years, case of chorionic gonadotropin therapy for dysmenorrhea. Shows condition of gingivae before chorionic gonadotropin therapy.

Fig. 39.—Same case as Fig. 38. Shows degenerative changes in the gingivae following administration of chorionic gonadotropin. Note tumefaction and inflammation of upper anterior papillae. Condition diagnosed as a subacute Vincent's infection involving chiefly the upper teeth on both labial and lingual aspects.

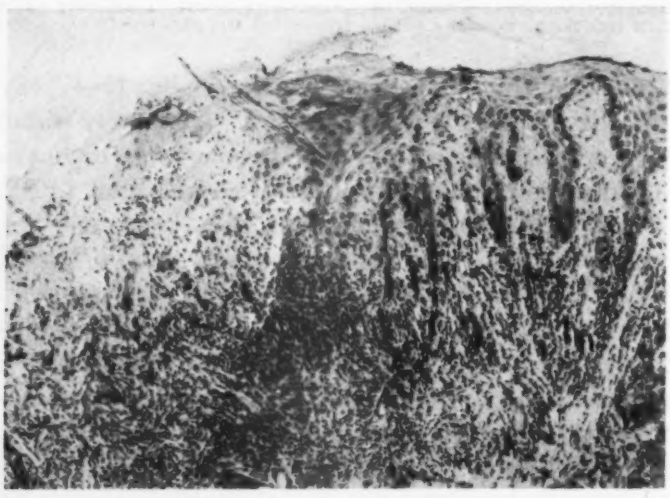


Fig. 40.—Same case as Fig. 39. Shows reduction in keratin, ulceration, and marked inflammatory infiltration following chorionic gonadotropin administration.

is not increased, the lack of it would tend to ameliorate an estrogen deficiency. However, the significance here as to gingival effect would depend on the proportional amounts present as influenced by dietary intake.

The vitamin C content of maternal plasma decreases as pregnancy advances when the intake is constant. However, when the vitamin C content of the maternal diet is increased, the serum ascorbic acid also increases (Teel, Burke, and Draper, 1938).



Fig. 41.—V. S., Negro girl, aged 9 years, case of thyroid dysfunction. Section of alveolar gingiva near tip of upper right cuspid papilla showing degenerative changes in the epithelium. Note the marked similarity of the section and the presence of hyaline bodies like those seen in Fig. 27. (Magnification ×215.)

THE ETIOLOGY OF PREGNANCY GINGIVITIS

The most prominent changes in the gingivae and oral mucous membranes during pregnancy are loss of surface keratin, hydropic alteration of the stratum spinosum, hyperplasia of the stratum germinativum and inflammatory changes in the lamina propria. Analogous changes have been observed in the vagina during pregnancy. Papanicolaou (1933) has shown that there is a great hypertrophy and hyperplasia of the vaginal epithelium during pregnancy, but that the normal nonnucleated cornified cells are seen less frequently as pregnancy advances and disappear almost entirely in late pregnancy.

The nasal mucosa is another epithelial surface which shows characteristic changes during pregnancy. Mortimer, Wright, and Collip (1936, 1937) have shown that as gestation progresses, the color of the nasal mucosa becomes increasingly red. The third month is the top limit of normal; from the fifth to the seventh month, the color is above normal and by the eighth month the nasal mucosa is very red and swollen. Therefore, the pregnancy changes found in the mouth are not specific for the oral mucous membranes, but also occur on epithelial surfaces elsewhere in the body.

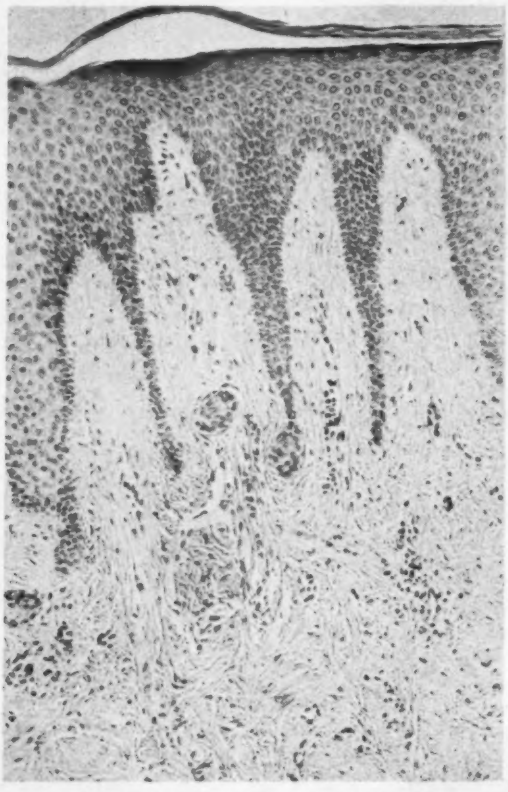


Fig. 42.—J. R., man, aged 29 years, case of Addison's disease. Shows increased keratinization of alveolar gingiva, epithelial hyperplasia with rete pegs penetrating deeply into the lamina propria, and a great increase in connective tissue. (Magnification ×160.)

The observed decrease in keratinization may be caused by an altered metabolism resulting in a subnormal estrogen effect on the tissues. Ziskin and Blackberg (1940) have shown that a lack of estrogen leads to degenerative changes in the gingivae and oral mucous membranes. In a female patient with hirsutism and secondary amenorrhea, there was loss of surface keratin (Fig. 29). Estrogen injection, on the other hand, causes a decrease in gingival inflammation and an increased thickness of surface keratin, as seen in Figs. 30 and 31. These figures illustrate the gingival changes in the monkey treated experimentally with estrogen for twenty-five days. The effect of stilbestrol on the gingivae is similar to that of the natural estrogens. The gingival changes in a patient under stilbestrol treatment for two years are shown in Figs. 32, 33, and 34. There was an increase in thickness and maturity of the surface keratin,

a downward growth of the epithelial pegs, and a great increase in thickness of the connective tissue of the lamina propria (hyperplasia).

The increased amount of progesterone found in pregnancy (Browne, Henry, and Venning, 1937) would also tend to alter the estrogen effect, since progesterone has a sparing action on estrogens, reducing catabolism and utilization (Smith and Smith, 1941). Also, Hall (1945) has shown that if the decrease in vaginal cornification during pregnancy (as described by Papanicolaou) does not occur, abortion or miscarriage may result; however, by administration of progesterone in an attempt to prevent miscarriage, the vaginal cornification is reduced, presumably by modifying estrogenic activity.

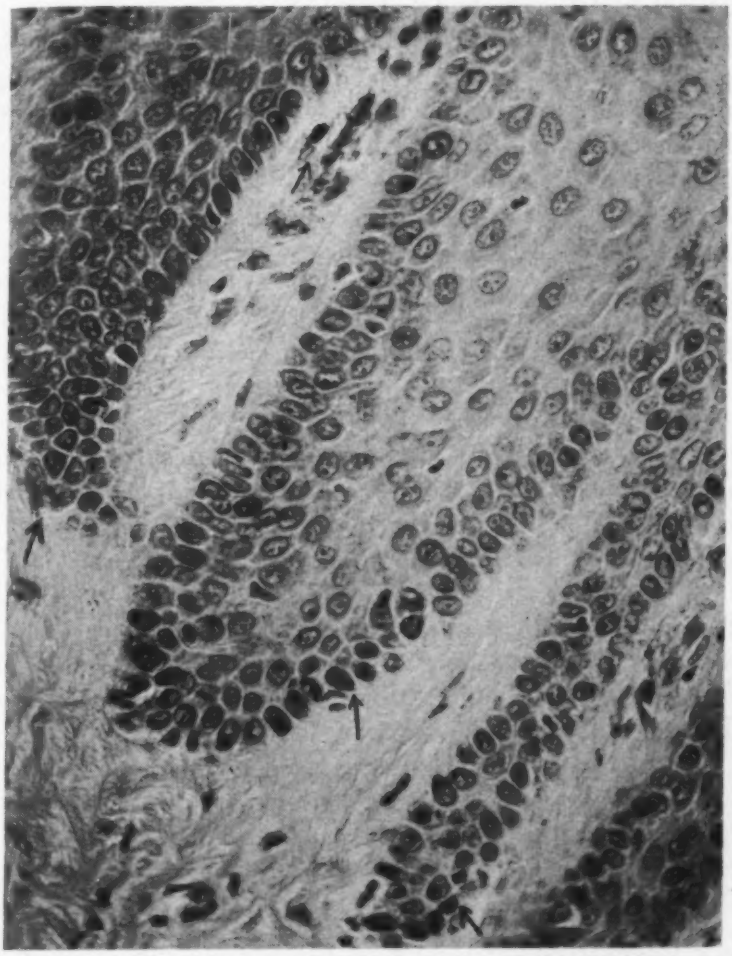


Fig. 43.—Higher power of Fig. 42. Note the melanin pigment in the basal cells of the epithelium and in the connective tissue at arrows. (Magnification ×594.)

Ziskin (1938) has shown that chorionic gonadotropin injections cause retrogressive changes in the epithelium of both castrates and noncastrates, the changes being more marked in the castrate. The gingival changes in a monkey treated experimentally with chorionic gonadotropin for sixty-eight days are shown in Figs. 35 and 36; following treatment, there was loss of surface keratin, parakeratosis, and a cellular hyperplasia. The inflammatory changes of the gingivae

of a monkey that had received chorionic gonadotropin injections for sixteen days are shown in Fig. 37. The gingival changes simulated subacute Vincent's infection. The changes in a patient's gingivae following the administration of chorionic gonadotropin for dysmenorrhea are shown in Figs. 38, 39 and 40; there was surface epithelial necrosis, and a marked inflammatory reaction in the lamina propria. Clinically, the condition was diagnosed as subacute Vincent's infection. Thus, the changes produced by chorionic gonadotropin injections, experimentally and clinically, are of the type seen in the gingivae during pregnancy. The large amount of chorionic gonadotropin found in the urine during pregnancy, the metabolic function of which is unknown, may, however, play some role in the epithelial alteration.

Evidence has been presented showing that the pituitary gland is in a hyperactive state during pregnancy (Rasmussen, 1933; Severinghaus, 1938, 1939). This pituitary hyperactivity may also account for the observed hyperplasia of the basal cell layer of the epithelium which occurs despite the keratin loss and hydropic degeneration of the stratum spinosum. Chorionic gonadotropin must also be considered as causative of this change, since epithelial hyperplasia was induced experimentally with a crude extract of pregnancy urine (Fig. 36).

There is also increased activity of the thyroid during pregnancy (Hughes, 1940). Since the action of thyroxin may be such as to produce a state of low estrogenic function (Lerman, 1942), this increased thyroid function may cause decreased estrogenic utilization. However, low estrogenic function may also result from hypothyroidism. A case of thyroid disturbance is shown in Fig. 41. The patient was hyperthyroid, having a basal metabolic rate of +34 per cent in August, 1938, just prior to a hemithyroidectomy. However, when the gingival biopsy was taken in June, 1941, the basal metabolic rate had fallen to -24 per cent. The changes seen are not typical of either hypothyroidism or hyperthyroidism. There was a reduction of surface keratin, degeneration of the stratum spinosum, and basal-cell hyperplasia. The changes in this case are similar to those seen in the case of pregnancy gingivitis shown in Figs. 24, 25, 26, 27, and 28. Of particular interest is the similarity between Fig. 27 and Fig. 41 (of comparable magnification), both of which are grossly similar, have analogous surface degeneration, hydropic alteration, and hyaline body formation in the stratum spinosum, and basal-cell hyperplasia.

The altered activity of the adrenal cortex during pregnancy may be another factor tending to modify the available estrogen. A case of Addison's disease showed an increased thickness and parakeratosis of the surface keratin, hyperplasia of the basal cell layer, and increased thickness of connective tissue in the lamina propria (Figs. 42 and 43). Conversely, in a patient with adrenal hyperplasia and pseudohermaphroditism, there was a marked reduction in the surface keratin, pyknosis and hydropic degeneration in the stratum spinosum, hyperplasia of the basal cell layer, and perivascular infiltration in the lamina propria (Figs. 44 and 45). (Compare also with case of hirsutism, Fig. 29.) Thus, hyperactivity of the adrenal cortex causes gingival changes analogous to those seen in pregnancy.

The altered physiology of pregnancy may cause changes in liver function. In the liver, during pregnancy, there is a disappearance of glycogen from the central portion of the lobule, reduced capacity for glycogen storage, a passive congestion of the central veins, and stagnation of bile in biliary passages;



Fig. 44.

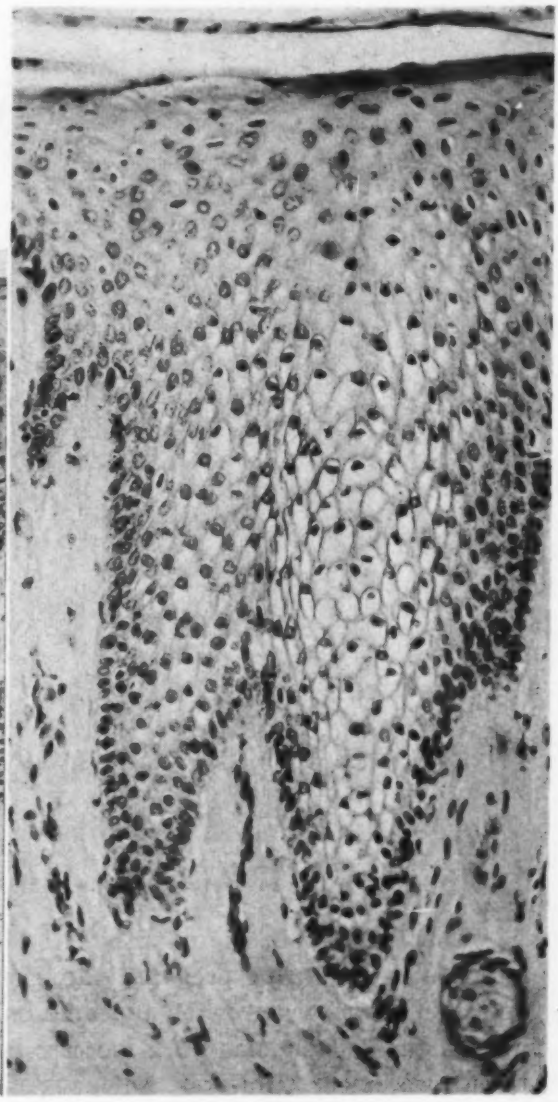


Fig. 45.

Fig. 44.—C. A., case of adrenal hyperplasia and pseudohermaphroditism. Aged 7; physical age was 25. Infantile ovaries and uterus removed. No testicular tissue was found. Hirsutism and enlarged clitoris gave appearance of male genitalia. Is being raised as a boy. Section of the alveolar gingiva showing reduction of the surface keratin and epithelial hydropic degeneration and hyperplasia. (Magnification ×178.)

Fig. 45.—Higher power of Fig. 44. (Magnification ×336.)

glycosuria may occur because of an abnormality of liver function. For comparison, we have included a case of cirrhosis of the liver in which there was a stratum lucidum-like parakeratosis of the cheek, and pigment deposition in the basal

cells (Figs. 46 and 47), indicating how altered liver function may result in changes in epithelial tissues. The decrease of stored glycogen and the presence of glycosuria in some cases may also account for some of the gingival changes during pregnancy.

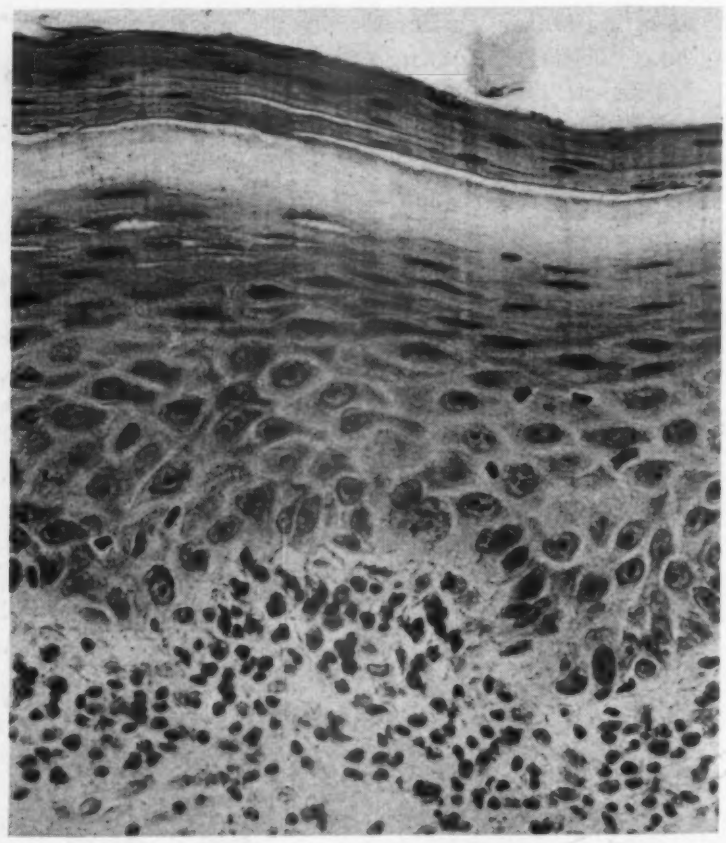


Fig. 46.—W. L., man, case of cirrhosis of liver. Section of right cheek showing stratum lucidum like parakeratosis of the epithelium. (Magnification ×660.)

There are changes in tissue reactivity during pregnancy. The changes within a nevus during pregnancy are shown in Figs. 48, 49, 50, and 51. During gestation, tumorous growths appeared on the left cheek and forehead within the area of the hemangioma. An increase in edema and vascularity of the gingival tissues was seen. However, following parturition, the growths on the face and gingivae disappeared without treatment and the edema diminished. A benign growth of the tongue during pregnancy is shown in Fig. 52. The tumor made its appearance during pregnancy and attained considerable size. Following parturition the growth regressed somewhat, but in contrast to the growth appearing in the nevus it did not disappear completely. It was excised and found microscopically to consist mainly of a vascular and cellular connective tissue.

During pregnancy, there is also an altered metabolic and nutritional balance. We have discussed some of the relationships between the vitamins

and the endocrine system. The alteration in nutritional status during pregnancy undoubtedly causes some change in the endocrine equilibrium.

The observed changes in the gingivae and oral mucous membranes are not specific and vary in different individuals. Thus, in one of the cases, there was an increase in keratinization rather than the decrease observed in the other cases (Fig. 38). The changes apparently are not the result of a single etiological factor, but rather they are a manifestation of the altered nutritional, and

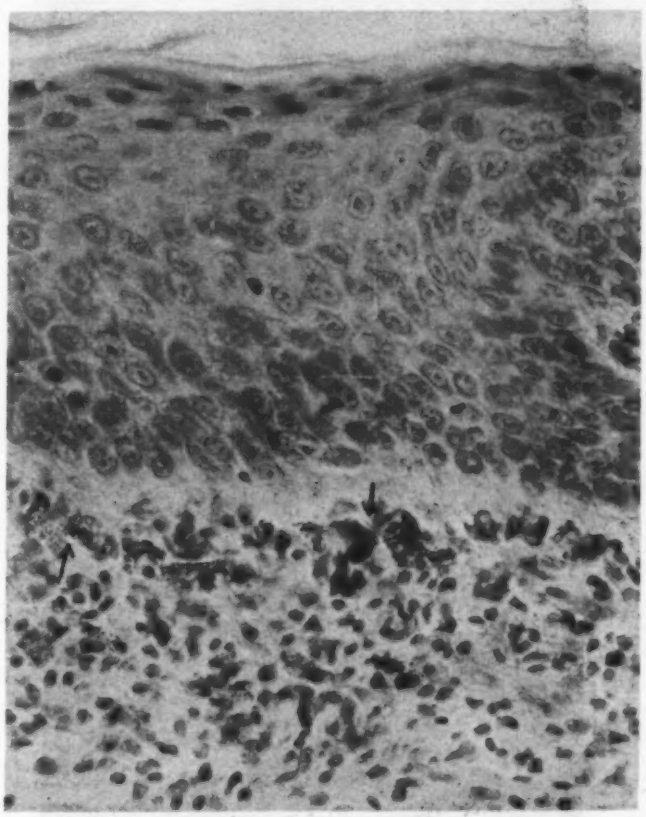


Fig. 47.—Same case as Fig. 46. Shows melanin pigmentation in basal cells and connective tissue. (Magnification ×660.)

metabolic status of the individual during pregnancy. Probably the most prominent cause of the gingival changes is a lack of utilization of estrogen or modification of endogenous estrogen. The mechanism involved may be explained in several ways:

1. The increase in serum and urinary estrogen does not have a proportional estrogen effect of the gingivae since part of the serum estrogen is bound to the protein fraction of the blood, while the urinary estrogen is excreted as the conjugated glucuronide which is a detoxification product.

2. There may be reduced ability of the oral tissues to utilize the available estrogen because of the increased amount of progesterone which has a sparing action on estrogen, modifying its utilization.

3. The large amount of chorionic gonadotropin found during pregnancy in some way affects the gingivae although the mechanism is unknown.

Fig. 48.



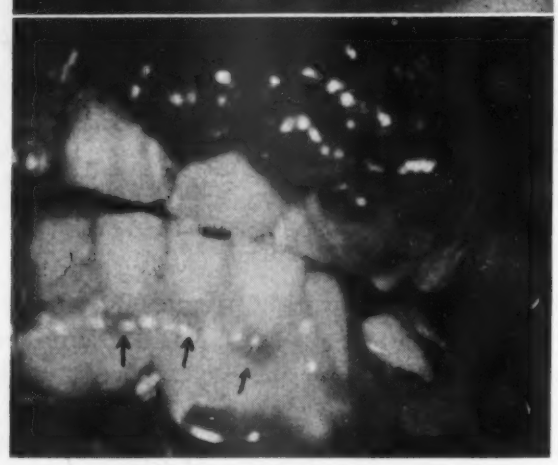


Fig. 49.

Fig. 48.—M. C., aged 27 years, white, primipara, seventh month. The dark area covering most of the left cheek is a nevus. In this area may be seen several pregnancy tumors which sprang up about the fifth month and were confined to area of the nevus.

Fig. 49.—Same case as Fig. 48. The gingivae and mucous membranes of the upper jaw on the left side are also involved in the nevus formation. However, there is marked tissue proliferation of the upper gingivae due to the pregnancy and new growths in the lower anterior region similar to those seen on the cheek in Fig. 48.

Fig. 50.





Fig. 51.

Fig. 50.—Same case as Fig. 48. Photograph taken three and one-half months later. "Pregnancy tumors" on face in area of nevus have disappeared without treatment following parturition.

Fig. 51.—Same case as Fig. 48. Edema and proliferation of gingivae are greatly reduced following parturition, although there is still some tissue overgrowth on the upper left side. The growths in region of lower anteriors have disappeared, also without treatment.

4. The increased activity of the thyroid gland and the adrenal cortex may also tend to decrease or modify the effect of the available estrogen to the gingivae.

5. Nutritional factors may alter the amount of endogenous estrogens available for the gingivae. Any or all of the above mechanisms may be involved in a particular individual during pregnancy. The end result is a lack of utilizable estrogen, which manifests itself in the gingivae and oral mucous membranes as pregnancy gingivitis.



Fig. 52.—L. A., aged 36 years, multipara. Shows tumor growing on the side of the tongue which appeared during fifth month and remained throughout pregnancy at about the same size. Following parturition it regressed slightly but did not disappear; it was excised and consisted of a vascular fibrous connective tissue.

SUMMARY

A comprehensive review of the history of pregnancy gingivitis is presented.

Histologic studies were made of the gingivae of a large group of pregnant women, of whom twenty are reported in table form. The biopsies were taken at different months during gestation from those women exhibiting clinical manifestations of pregnancy gingivitis. Clinical photographs were also taken and are included.

Histologic studies were made of the gingivae of patients with endocrine disturbances and of monkeys treated experimentally with hormones. Photomicrographs and clinical photographs of representative cases are included.

A clinical classification of pregnancy gingivitis, representing the progressive changes in pregnancy, is presented.

The most prominent changes in the gingivae and oral mucous membranes during pregnancy were a loss of surface keratin, hydropic alteration of the stratum spinosum, hyperplasia of the stratum germinativum, and inflammatory changes in the lamina propria.

A comprehensive review of endocrine and metabolic relationships is presented. This includes a consideration of the action of the hypophysis, the thyroid gland, the adrenal cortex, the ovaries, the placenta, and the vitamins.

The histologic changes apparently do not arise from a single etiological factor. Rather they are a manifestation of the altered nutritional and the metabolic status of the individual during pregnancy. However, the immediate cause of the gingival changes appears to be a diminished utilization of estrogen or a modification of estrogen metabolism. The mechanism involved may be explained in several ways: (1) Estrogen during pregnancy is present in bound, inactive form, so that it may not be available to the gingivae to the expected extent, and hence does not have a proportional effect on the gingivae. (2) There may be a reduced ability of the oral tissues to utilize the available estrogen because of the increased amounts and modifying effect of progesterone. (3) The large amounts of chorionic gonadotropin may affect the gingivae directly or indirectly. (4) The increased activity of the thyroid gland and the adrenal cortex may modify the effect of available estrogen on the gingivae. (5) Nutritional factors may alter the amount of endogenous estrogens available for the gingivae. These factors indicate the probable causes of the gingival alterations reported here.

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Original Articles

ANESTHETIC MANAGEMENT IN RECONSTRUCTIVE SURGERY OF THE MANDIBLE

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OPERATIVE procedures upon the mandible pose special problems in anesthesia in direct proportion to the complexity of the surgical manipulations. Replacement of mandibular defects by bone grafting presents a difficult situation for surgeon and anesthetist alike. It is proposed, in this discussion, to elaborate upon the principles concerned in anesthetic management and to describe the results obtained in a series of 67 patients whose mandibular disabilities were traumatic in origin.

The experience of others in this field provided only indirect assistance in the evolution of a satisfactory approach to anesthesia for these operations, since most of the work reported was concerned with the fixation of mandibular fractures or the treatment of acute injuries to the face and lower jaw. Few data pertaining to anesthesia for extensive plastic repair of the mandible are available.

Local or regional anesthesia is advocated by some[1-4] as the method of choice for surgical adjustments of the mandible. The objections to general anesthesia deal largely with the hazards of respiratory obstruction during operation and those resulting from emesis postoperatively.

On the other hand, other authors have been favorably impressed by the utility of general narcosis for the comfort of the patient and the surgeon.[5-7] The latter group has emphasized very properly the importance of unobstructed respirations secured by the employment of an intratracheal airway with ether, the most commonly used anesthetic agent.

Quite early in the consideration of the problems encountered, it was decided that regional anesthesia was not the method of choice for bone grafts to the mandible. The inconvenience, and occasionally the impracticability, of anesthetizing the donor sites (rib, tibia, and iliac crest) for the grafts, in addition to the mandibular area was objectionable. Second, infiltration of local anesthetic agents into the operative site was a possible source of infection, a disastrous sequela in bone-grafting procedures. Third, nerve blocks proper were not always technically possible because of concomitant injuries to the head and face. Fourth, the length of operation (three to five hours) in a posture that necessitated complete immobility of the jaw imposed too great

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a burden upon the conscious patient. Finally, the period of analgesia afforded by nerve block was usually inadequate for the completion of the surgical task, and repetition of a block during the operation was unsatisfactory and introduced a source of contamination of the operative field. For all these reasons, local and regional anesthesia were not considered the anesthetic methods of choice for the type of operative procedure under discussion.

Some form of general anesthesia was necessary, therefore, in the proper anesthetic management of these patients. In the light of past experience and present knowledge about the hazards of operating in or near the anatomical airway without adequate control of respiratory activity, intratracheal technique was absolutely essential. The need for an intratracheal catheter passed transnasally was further emphasized by the requirements of the surgeon that as little anesthesia apparatus as possible be near the operative field to minimize the possibility of infection of the grafted areas and to facilitate convenient operating conditions. Once the desired technique of nasotracheal anesthesia had been decided upon, many difficulties remained as considerable hurdles to be surmounted.

Anatomical distortion of the nose, mouth, face, and oral contents was not infrequent and often made intubation a serious mechanical problem. To gain the maximum benefit of being prewarned of difficulty ahead, patients who "looked difficult" in the course of the preoperative review were referred to the otologist for complete examination of the nose, nasopharynx, oropharynx, and larynx. This examination was done in the presence of a member of the anesthesia section and the anatomical abnormalities of the upper respiratory passages carefully noted. This information proved invaluable in the cases studied and facilitated more than one allegedly impossible intubation by the selection of the correct size of intratracheal tube and its careful insertion with due regard to the anatomical obstacles present.

Another difficulty for the anesthetist was the presence of mandibular splints devised especially for the surgical management of these cases. These splints[8] were important aids to the surgeon but they encroached on the oral aperture to a fairly considerable degree and made visualization of the glottis more difficult than is usually the case. The loss of oral spread was further aggravated by the results of trauma which, in itself, limited the ability of the patient to open his mouth widely.

It may be argued by some that blind nasal intubation either in the conscious state or after the induction of anesthesia can obviate the handicaps described, and the argument has a certain validity. However, it must be remembered that the pathway for the tube is cicatricial and of poor quality in these patients. Failure at intubation or the slightest departure from scrupulous gentleness in maneuver during blind intubation frequently resulted in hemorrhage and necessitated cancellation of the operation. Therefore, blind intubation was abandoned and direct visualization of the glottis was accomplished with the MacIntosh laryngoscope. This instrument was selected because of its utility in passing through a narrow oral aperture and the ex-

cellent visualization rendered with a minimum of trauma. Intubation was completed very cautiously and gently under direct observation. With the nasotracheal tube in place, the conduct of the anesthesia during the surgical procedure was not particularly difficult. Operations upon the mandible and adjoining tissues do not require relaxation and the requirements were met satisfactorily by anesthesia of a depth sufficient to permit toleration of the intratracheal catheter. In fact, muscular relaxation was avoided during operation, and firm fixation of the mandible to the maxilla was effected with wires.

An important difficulty arose in the immediate postoperative period after the intratracheal airway was removed. At the conclusion of the operation, to preclude slipping of the bone grafts in the mandible it was necessary to maintain the wire fixation described above. The pharynx and lower air passages became poorly accessible for the aspiration of mucus, the removal of vomitus, the control of respiration, and the performance of tracheobronchial aspiration. The management of these complications required the removal of the fixation wires endangering the position and subsequent viability of the graft.

The first 14 anesthesias were performed in a reasonably classical manner in that nitrous oxide-ether sequence was employed and intratracheal intubation performed either blind or by direct vision. Morphine and scopolamine in the ratio of 25 to 1 were administered preoperatively. A closed system with circle filter carbon dioxide absorption was maintained with the aid of a pharyngeal pack of moistened gauze. It soon became apparent that the desiderata were poorly accomplished. Emesis was frequent (50 per cent) (Table I), and much mucus was present postoperatively, severe enough to produce cyanosis in many of the patients and quite difficult to remove by suction because of the wired jaws.

TABLE I

ANESTHETIC AGENTS	NUMBER OF CASES	POSTOPERATIVE RESPIRATORY OBSTRUCTION (PER CENT)	ATELECTASIS (PER CENT)	EMESIS (PER CENT)
Nitrous oxide, oxygen, curare	39	0	0	17
Ether, nitrous oxide	14	36	14	50
Ether, nitrous oxide and scopolamine postoperatively	10	10	0	40
Pentothal, nitrous oxide, cocaine spray	4	0	0	50

Atelectasis occurred in two of these fourteen patients on the first day. Treatment was most difficult, and relief was obtained by conservative means of encouraging cough, frequent turning, and deep breathing. Another patient narrowly escaped an obstructive death with mucus and vomitus. Resuscitation involved removal of the wire fixation to aspirate the detritus which was responsible for the obstruction and caused eventual loss of the graft.

Thus, it was apparent that the anesthetic management left much to be desired, particularly during the postoperative period. The first thought by

way of improvement was the use of pentothal sodium instead of ether. Despite the fact that intratracheal intubation can be done during pentothal anesthesia, there is considerable danger of laryngospasm and asphyxia. This point was emphasized by Thornton and Rowbotham[9] in the discussion of the anesthesia problems in acute injury to the face and jaws. Another objection to pentothal was the possibility of laryngospasm during extubation. Finally, there was no real assurance of prompt postoperative recovery even with skillful use of the drug.

Pentothal was employed in four instances following cocaine spray. Mucus and emesis still remained important complications in the postoperative period. Therefore, the effect of depressing the patient's oral and tracheobronchial secretions for the first twelve hours after operation by the subcutaneous injection of small doses of scopolamine (0.0002 Gm.) every three hours, beginning three hours after the preoperative dose, was studied. There was definite improvement in the results in the ten patients so treated, in that obstruction due to mucus occurred but once. However, emesis was still a problem and occurred frequently (40 per cent) (Table I). Another unpleasant complication was the loss of cortical inhibition following the scopolamine series, manifested in some patients by delirium and hyperkinetic activity.

At this time, it became evident that the entire situation could be well handled if a nasotracheal airway were introduced without trauma, anesthesia maintained without hypoxia, and there was a prompt return to consciousness at the completion of the operation. Despite wire fixation, a conscious patient is well protected in many ways against respiratory obstruction. He can cough, expectorate, or, if necessary, swallow any material in his mouth without endangering the bone graft.

The accomplishment of these aims was provided for by the employment of nitrous oxide anesthesia and the administration of curare for the sole purpose of securing maximal relaxation of the jaw muscles for intratracheal intubation. In actual practice, the cases were managed in this manner. Premedication consisted of a barbiturate (seconal or nembutal) and morphine two hours prior to induction, followed by a repeated dose of the two drugs with scopolamine one hour later. Dosage was estimated to produce a patient that was drowsy and confused at the inception of anesthesia but not excessively depressed. Proper premedication is of great importance in employing this anesthetic regime.

Induction was then effected with nitrous oxide-oxygen in 80:20 mixture by semiclosed technique. At no time was the oxygen concentration allowed to fall below 20 per cent.[10] When surgical anesthesia was reached, usually first plane, inflation of the lungs was performed. After checking airway control in this manner, curare was given intravenously in doses varying from 60 to 120 mg. depending on the patient's size. As soon as the muscles of the head and neck were relaxed (two to ten minutes), transnasal intratracheal intubation was accomplished under direct vision, employing the MacIntosh laryngoscope. Usually intubation was not difficult, but it must be accomplished rapidly because of the rapid recovery from nitrous oxide anesthesia.

A small amount of ether vapor is needed at this time in some cases. Occasionally, there is partial cord adduction after curarization, but gentle pressure upon the intratracheal catheter readily breaks through this resistance. The dose of curare used always depresses respiration, but there has been only one instance of respiratory arrest.

After intubation was completed, nitrous oxide anesthesia was resumed in concentrations varying from 50 per cent to a maximum of 70 per cent. It should be pointed out that there is mild "bucking" on the tube immediately after intubation before anesthesia is resumed in some cases.

The pharynx was packed off with moist gauze to maintain a closed system. The dental surgeon then wired the jaws together, a procedure greatly facilitated by the relaxation provided by curare.

The maintenance of nitrous oxide-oxygen anesthesia during the operation required attention to a few important details. The concentration of nitrous oxide was never greater than 70 per cent after the completion of the induction, and the breathing bag was emptied periodically to assure the elimination of accumulated exhaled nitrogen. If satisfactory anesthesia could not be maintained with a minimum of 30 per cent oxygen, small doses of morphine were given intravenously to reinforce the nitrous oxide-oxygen mixture.

At the completion of operation, the gauze packs were removed without disturbing the fixation wires if possible. If it was necessary to remove the wires temporarily, the mandible was moved very carefully by the oral surgeon. The wires were immediately reapplied, and 20 to 60 mg. curare given if reposition of the jaw was difficult. Because of the latter group of cases, an endotracheal tube with an inflatable cuff is preferable to pharyngeal packs to obviate the hazard to the graft of removing the wires at the termination of operation. This tube must be different from the usual cuffed tube in that its cuff and catheter occupy no more room than the tube itself in passage through the nose.

The intratracheal catheter was removed when the patient was restored to consciousness after several flushings with oxygen. Emesis was lessened (17 per cent) (Table I), and severe in only one patient. No other complications of note have occurred. The only postoperative difficulties encountered occurred when opiates were administered in excessive dosage. Nursing care has been greatly simplified in that a fully conscious patient is less likely to require suction and close observation.

Thus far, 39 patients subjected to bone grafting of the mandible have been anesthetized with nasotracheal nitrous oxide-oxygen with intubation facilitated by curare.

The excellent results obtained have made the method most useful for bone grafts to the lower jaw and have suggested its use in other situations where prompt restoration of consciousness is advantageous, or where emesis and the resultant increased venous pressure threaten the viability of difficult tube grafts about the head and neck. Thus far, only meager experience has been acquired with this technique in soft-tissue plastic repair of the face, head, and neck, but the results are encouraging.

SUMMARY

1. Some of the problems in anesthesia occasioned by reconstructive surgery of the mandible are discussed.

2. Sixty-seven cases of bone grafting to the mandible form the basis for this discussion. Thirty-nine were anesthetized with nitrous oxide-oxygen anesthesia aided by curare; fourteen with ether-nitrous oxide sequence; ten with ether, nitrous oxide, followed by scopolamine in the postoperative period; and four with pentothal (cocaine topical). All were intratracheal.

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CONGENITAL MANDIBULAR DEFORMITIES IN NEWBORN INFANTS

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CONGENITAL mandibular deformities in newborn infants have been frequently found, but infrequently described.

Chapple described this deformity in the pediatric literature, but apparently there have been no reports of these conditions as a result of intrauterine pressure in the dental literature.

Because of the possible confusion between this picture, and actual dislocation or fracture of the mandible, we feel justified in presenting this small series of cases to illustrate how mandibular deformities might occur.

Our particular interest in congenital mandibular deformities became aroused recently when we were confronted with a newborn infant with marked deviation of the mandible. The obstetrician gave a history of applying considerable force to the mandible during the process of delivery, and it was felt that this infant had a dislocation or fracture of this bone. X-ray studies failed to exhibit either condition, although radiographic examination was made somewhat difficult because of the lack of any roentgenograms of either the temporomandibular joints or the mandibles of one-day-old infants (Figs. 1 and 2). Clinical examination revealed fullness of right side of face, and a marked lateral excursion of the mandible to the right. The patient was in no distress, and there was no tenderness evident. The jaw could be moved freely, and when the mouth was opened widely, the deviation became more prominent. The infant could nurse without any particular distress and had no difficulty in swallowing. There was no paralysis of the muscles of mastication and no evidence of a fifth cranial nerve lesion. Further examination demonstrated a "depression" of the left side of the face, and it was assumed that this deformity was due to intrauterine pressure, with possibly one of the hands or feet fitting into this "depression."

Careful oral examination was made of the sixty newborn infants delivered during the next month, and it was found that twelve of them had deviated mandibles. All of these deliveries were essentially normal, and the obstetricians could offer no history of pressure applied to the mandibles. In each case, there was a variation of the mandible from the normal horizontal plane, as well as to the right or left, with the greatest distance between the maxilla and mandible occurring on the side toward which the mandible was deviated. In all cases a hand or foot could be placed in juxtaposition to the indented defect of the mandible so that it "fitted" the deformity. All the babies had normal mandibular movements with satisfactory right and left lateral excursion. There was no difficulty in nursing or feeding.

The opinions or assertations contained herein are the private ones of the writers and not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.



Fig. 1.—Roentgenogram of normal right mandible of one-day-old infant.



Fig. 2.—Roentgenogram of normal left mandible of one-day-old infant.

A pair of identical twins were delivered under essentially normal conditions. One of them, *A*, had a normal maxillomandibular relationship, while *B* had a marked mandibular deformity with deviation to the left (Fig. 3). We could not be sure if this deviation was produced by the patient's own intrauterine position, or by pressure from the other twin (Fig. 4). However, these findings tend to indicate that the deformities are not due to any genetic disturbance, but are more likely due to intrauterine pressure. No other congenital deformities were noted in any of these cases, except in one case where a heart murmur was heard at a later checkup. Photographs were taken in an effort to



Fig. 3.—Identical twins. Baby A has normal maxillomandibular relationship. Baby B has mandibular deformity with deviation to left. Inclination of mandible from horizontal is also demonstrated.



Fig. 4.—Indicating possible position of comfort, with right foot placed under right mandible. It is possible that this deformity in one twin was caused by the pressure of some part of the other.

demonstrate the altered facial contour and mandibular deviation, and to duplicate the positions of comfort.



Fig. 5.—Deviation of mandible to left.



Fig. 6.—Apparent position of comfort of same baby as in Fig. 5.

These deviations are characterized by a malrelationship of the maxilla and mandible, and an alteration of the normal facial contour. These malformations may be so marked that they may be mistaken for congenital dislocation or fracture.

Chapple has described these congenital mandibular deformities, and offers an excellent explanation for their cause. He feels that they are caused by an abnormal intrauterine position and pressure. Often a hand or foot may be held by the mandible to the opposite side. During the first week of life, it is often possible to place these infants in a "position of comfort." In this position, the



Fig. 7.—Deformity of mandible to right and deviation from horizontal plane.



Fig. 8.—Same baby as in Fig. 7. Position of comfort with right hand under left mandible.

limb can be placed against the face with ease and comfort, and it is Chapple's impression that these positions of comfort were maintained during the latter months of fetal life. There are many positions of comfort, and their reconstruction of the fetal position often explains the mechanism of clubfoot, torticollis, brachygnathia, asymmetry of face, dislocations of wrists, elbows, knees, and hips (Figs. 6 and 8). Thus, when deviation of the mandible is present, it is very important to look for other defects, especially congenital dislocation of hips. Early recognition and treatment of these other defects are very important and the presence of a deviated jaw should never be overlooked.

Chapple stated that about 30 per cent of the newborn infants have some deviation of the jaw, whereas only about 20 per cent of our series of infants had deviated jaws.

Since none of these infants had any particular difficulties in nursing, no immediate treatment was deemed necessary. However, it is the opinion of one of the authors (R. E. S.) that if newborn infants are permitted to sleep on their abdomens and faces, for several months, there will result a marked narrowing of the face and flattening of the chest. The narrowing of the face results in a high sharp maxillary arch, receding of lower jaw, and overlapping of the upper teeth. This deformity of the face can be corrected if the infant is allowed to sleep on his back during the greater part of the first year.

Because of these impressions, it was felt that it would be undesirable to allow the babies to sleep in such a manner that increased pressure would be placed against the depressed aspect of the mandible, thus causing a greater deformity. For these reasons the babies slept on their backs, thus allowing the jaws to return to a normal position. Some of the milder deformities showed improvement within two weeks, whereas the severer deformities took about eight to ten weeks to assume a normal position.

SUMMARY

1. Congenital mandibular deviations must be differentiated from congenital dislocations or fractures.

2. Deviation of the jaw was found to be present in about 20 per cent of our newborn infants.

3. The deformities are due to intrauterine pressure, and not to a genetic disturbance, since only one of identical twins had this defect.

4. By reconstructing the position of comfort, it is often possible to illustrate the possible position in utero, and it is important to search for other congenital defects which might require early treatment.

5. In all cases, the deformities caused no difficulty in nursing or feeding and they were corrected spontaneously by the end of the tenth week.

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OSTEOTOMY FOR THE CORRECTION OF MANDIBULAR PROTRUSION

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MANDIBULAR protrusion or prognathism is a source of distress to those afflicted. They not infrequently suffer personality difficulties as a result of self-consciousness about this deformity. Kazanjian, Dingman, and others have noted an inferiority complex associated with this facial disfigurement. It is generally agreed that correction of this defect may result in a complete change of personality, and the patients become much better adjusted to their environment. Although such gains alone would fully justify surgical correction, there are other good reasons for such procedure.

The etiology of mandibular prognathism is obscure. Kazanjian has pointed out that heredity and/or disease may be a factor in the etiology of this condition. It is well known that acromegaly causes an abnormal growth of the mandible. An operation for correction of prognathism is not usually considered in a true case of acromegaly. Possibly some other type of hormonal disturbance may cause the mandible alone to grow abnormally. Abnormal forces of occlusion may stimulate the growth of the mandible. We have noted a geographical factor in the occurrence of prognathism. There seems to be a greater incidence of this condition in New England than in other parts of the country. This, of course, could be attributed to heredity.

In children, malformations of the jaws, as a rule, can be corrected by orthodontic measures. Osteotomy for correction of prognathism is only suggested for those patients in whom orthodontic treatment is impractical. Osteotomy is the only means of correction after full mandibular growth has been attained. It is well to bear in mind that the older the patient, the less favorable is the outlook for obtaining a satisfactory result by surgery.

Young adults with prognathism and with natural teeth usually are able to masticate their food only to a small degree. One patient that was operated upon at our clinic had only slight contact of the mandibular premolars with the maxillary cuspids. No other teeth occluded. Another patient had no articulating teeth.

Most authors report that their patients are fairly well nourished, and their principal complaint is their abnormal appearance. Later, as the patients begin to lose their teeth due to malocclusion, they complain of gastric symptoms, loss of weight, and nervousness. Their symptoms are not unlike those of incipient gastric ulcer. When these patients become edentulous, however, it is almost impossible to construct satisfactory full dentures. They then become total dental cripples with no hope of salvage by the prosthodontist. For this

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reason it is well to consider osteotomy for correction of prognathism while the patient is blessed with sufficient teeth to facilitate adequate fixation. This will help to issue a successful result.

The treatment of prognathism of the mandible by surgery is not new. In 1848, Hullihen successfully devised and performed an osteoplastic resection of a portion of the mandible. It was not until 1897, when Angle suggested bilateral partial osteoectomy, that the operation was considered again. The following year, Blair performed the second operation for this condition. Since that time many cases have been reported.

Many positions of the mandible and various degrees of prognathism are seen. There is the protrusion in which the deformity would seem to be caused by a simple elongation of the rami. There is a widening of the angle of the mandible until the mandible approaches a straight line drawn from the condyle through the ramus and the body to the symphysis. In this type, good occlusion can be obtained by sliding the mandibular arch posteriorly. A second type of deformity appears to result from an overgrowth of only the body of the mandible. There are also combinations of these two conditions. A third deformity is the typical open-bite, with occlusion only in the third molar region.

Numerous techniques have been advocated for the correction of these conditions. The nature of the deformity must influence the selection of the technique. For the simple elongation of the rami, Babcock, in 1909, suggested a bilateral section of the rami on a line anterior and posterior above the mandibular foramen and below the sigmoid notch. The mandible was then moved posteriorly and fixed in the desired position for bony union. Blair, in 1898, following the suggestion of Angle, removed a fragment from both sides of the mandible and corrected an overgrowth of the body. The anterior segment was moved posteriorly and fixed in apposition with the posterior fragments. He used interdental splints and elastic headbands to a chin cup as the method of fixation.

It is generally agreed that cases of open-bite can be corrected only by bilateral osteoectomy of a portion of the body of the mandible. Thoma advocates the removal of a Y-shaped fragment from the body. The anterior fragment can then be rocked upward and fixed with the fragments in maximum contact.

The distance that the mandibular arch has to be moved posteriorly in simple prognathism can be measured. If this distance is more than two-thirds the width of the ramus, an osteoectomy of the body of the mandible should be the operation of choice. A simple section of the rami should not be attempted because there will be insufficient contact of the fragments to insure strong union. This is important because of the very strong muscular pull on the condyloid-coronoid fragments by the temporal and external pterygoid muscles. The body and lower portion of the rami are pulled by the buccinator, masseter, and internal pterygoid muscle. This abnormal stress may result in a gradual opening of the bite in the anterior region. It may even result in a subsequent pathologic fracture of the new bony union. Excessive posterior movement of the body and lower ramus may cause the mandible to impinge on the mastoid process. This will inhibit proper function. There may also be a tearing or stretching of the mandibular

nerve with temporary or permanent anesthesia. These contraindications, if foreseen, will demand that a bilateral osteoectomy be performed on the body of the mandible rather than an osteotomy of the rami.

Prognathism resulting in excessive growth of the body of the mandible will be recognized by the wide spacing of the teeth. The rami and the angle of the mandible will be fairly normal. In this condition a bilateral osteoectomy of a portion of the mandible will be the technique of choice.

The simple protrusion which is caused by an elongation of the rami and in which normal occlusion can be obtained by simply sliding the body of the mandible posterior can easily be treated by a bilateral section of the rami. This is the technique used in the cases which we are presenting.

The means of fixation must first be determined. If sufficient teeth are present, fixation may be obtained by the use of intermaxillary wiring and the eyelet wiring method. A wire loop should be placed in every interproximal space, and each loop must be separate. The continuous Stout method of wiring should not be used because of the possibility of breaking one of the loops. Fixation can also be obtained by using orthodontic bands with small hooks for wires or rubber bands or by labial arch appliances. The Winter arch band could also be used with rubber-band traction. In two of our cases the eyelet wiring method was used. An eyelet was placed in every interproximal space. Both cases were successful. We used 0.01 stainless steel wire, and no wires were broken. In order to resist the forward thrust and the tendency for open-bites, interdental wires were applied diagonally from the upper second and first molar loops to the lower premolar and cuspid loops. The loops in direct apposition were also wires.

An incision 2 cm. long was made posterior to the ramus and below the lobe of the ear. By blunt dissection the ramus was exposed. It is imperative to identify and reflect upward the parotid gland together with the branches of the facial nerve. Injury to this nerve can result in permanent disability.

The method of sectioning of the rami was that suggested by Kazanjian. The lateral plate and the posterior and anterior border of the ramus were cut with a 702 crosscut fissure burr. The cut was made anteroposterior midway between the mandibular foramen and the sigmoid notch. The line of section should be slightly inclined downward from anterior to posterior. The inner plate can easily be fractured with a bone chisel or osteotome placed in the burr cut. Kazanjian recommends reflecting one of the coronoid-condyloid fragments medially and the other laterally. The upper and lower fragments may be wired together with stainless steel wire, or they may be allowed to assume a position determined by the pressure of the surrounding tissues. Dingman stated that he found it necessary to wire the upper and lower fragments at the time of operation. Three of his six cases not wired resulted in a nonunion. This occurred on one side in one of our cases. To correct the nonunion Dingman had to perform an open reduction and wire the fragments. It must not be forgotten, however, that a wire remaining in the bone is a foreign body. After reduction the wound is dusted with sulfanilamide powder and closed in layers without drainage. Triple "0" plain catgut was used to ligate all blood vessels and to approximate

the soft tissue. Interrupted silk sutures were used to close the skin. We wish to emphasize the importance of absolute hemostasis as an aid in bone union and the prevention of infection.

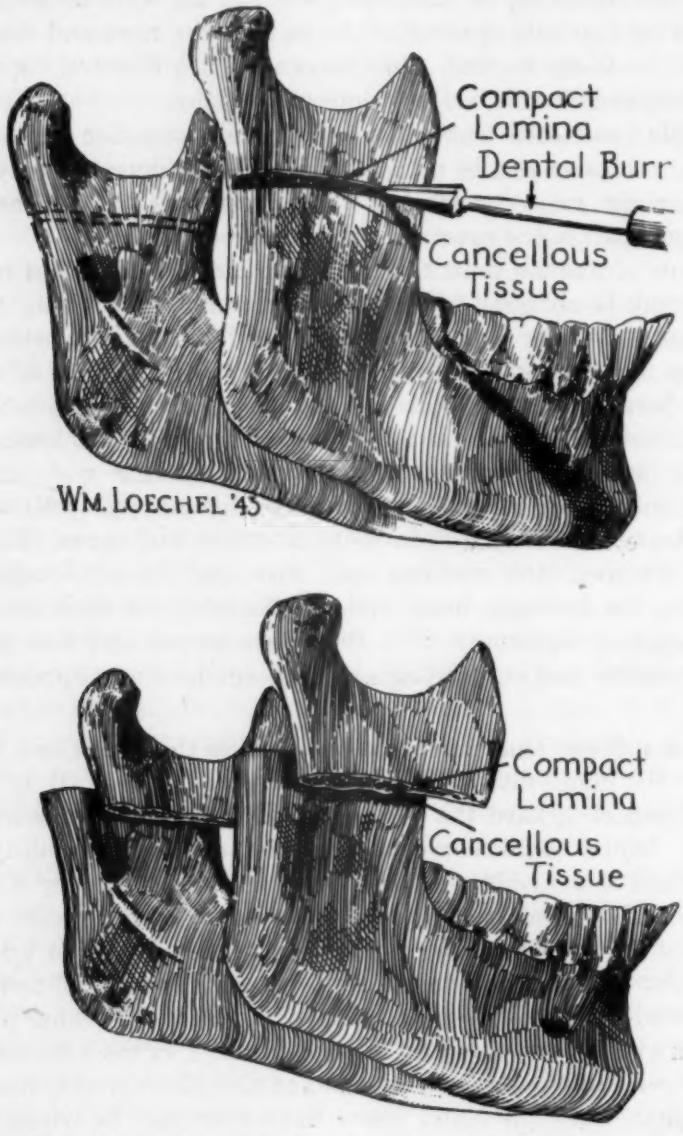


Fig. 1.—A diagrammatic drawing showing the approximate location of the burr cut and the position of the rami after the posterior movement of the body of the mandible.

Our patients were hospitalized and given a complete physical examination with special attention to their oral hygiene and to the cardiovascular and pulmonary systems. Laboratory studies included blood count, hemoglobin, bleeding and clotting time, serology, and urinalysis. Anteroposterior and lateral x-rays of the jaws were made preoperatively. It is advisable to take a full-mouth dental x-ray so that any emergency dental pathology can be corrected before the jaws are fixed together.

The maxillofacial examination must be complete. Study models should be taken of the two arches and mounted in their pathologic relationship. Photographic studies including pictures of the profile and of the relationship of the teeth in occlusion are taken. A careful study of the casts of the jaws and x-rays will reveal the amount of correction necessary. It will also reveal the type of occlusion to be expected. It may be necessary to grind one or several teeth before operation to facilitate a firmer intercuspal relationship.

Preoperative chemotherapy is advisable. We give 2 Gm. of sulfadiazine as an initial dose the day before surgery. This is followed by 1 Gm. every six hours. It is continued postoperatively until all danger of infection is past and the patient's temperature remains normal. The free administration (minimum 2,500 c.c. daily) of fluids is essential to guard against the formation of sulfa calculi in the kidneys.

We prefer pentothal sodium anesthesia with an intratracheal catheter inserted through the nose. Since pentothal sodium is given intravenously in the arm or leg, the anesthetist is out of the field of operation. The intratracheal catheter assures a free airway at all times. As preoperative medication, $\frac{1}{150}$ grain of atropine sulfate and $\frac{1}{4}$ grain of morphine sulfate were administered.

The postoperative care of these patients is similar to that of a compound fracture. Icecaps are applied to the jaws until edema has subsided. Fluids and chemotherapy are given the first postoperative day. Vitamin K is given to facilitate the blood clotting.

It is advantageous to keep a mechanical aspirator with a rubber catheter near the patient until he is well out of the anesthesia. It may be necessary to aspirate accumulations of mucus from the mouth and pharynx.

A review of our three cases may illustrate other points:

Case 1.—(Figs. 2 and 3.) G. H., a white man, aged 21 years, stated that as long as he could remember he had had a prominent lower jaw. He also had had trouble in biting and chewing his food. He thought this was the cause of his inability to gain any weight in the previous five years.

The technique used was the one suggested by Kazanjian. The operation was performed. The convalescence was uneventful, and the wires were removed two months later. The occlusion was very good. There was, however, a slight drifting forward to almost an end-to-end bite from the original assumed corrected position. There was no opening of the bite.

Case 2 (Figs. 4 to 7.) L. C., a white man, aged 32 years, was admitted to the medical service. He complained of a sharp pain along the edge of his ribs. It was the impression on admission that he had a peptic ulcer or gall bladder disease. His symptoms disappeared with medical treatment. It was noted during examination that he had a marked protrusion of the mandible, and the patient was wearing an unsatisfactory upper denture. The internist reasoned that his general condition, gastric symptoms, and underweight were due to his inability to masticate his food.

An osteotomy to improve his occlusion was recommended and performed. Because the patient was edentulous in the maxilla, a splint was constructed of

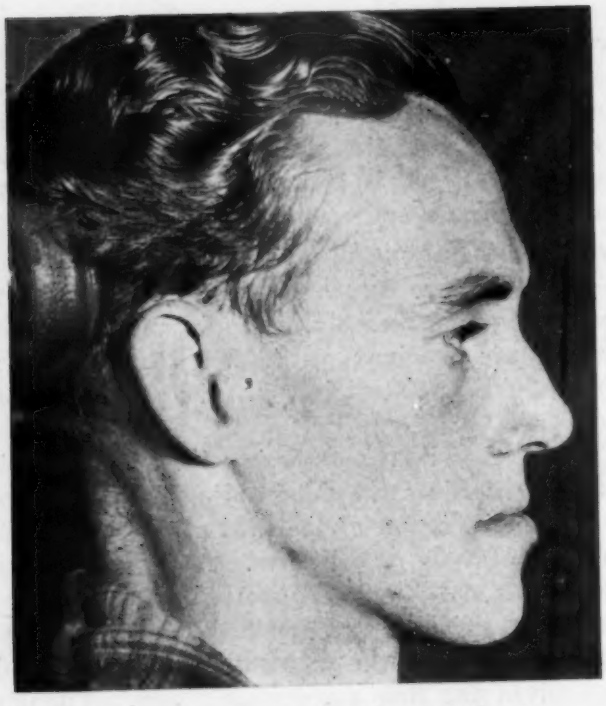


Fig. 2.—Case 1. Profile before osteotomy.

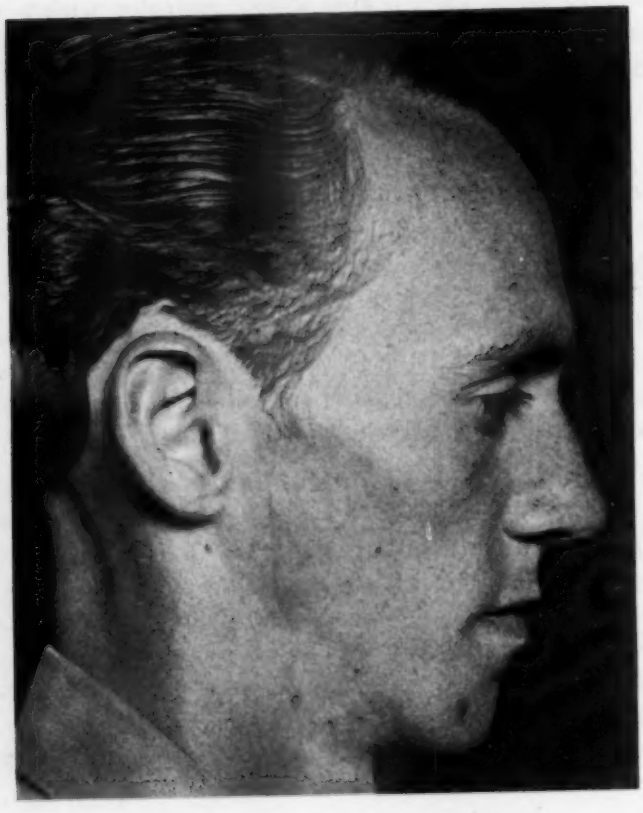


Fig. 3.—Case 1. Profile after osteotomy.

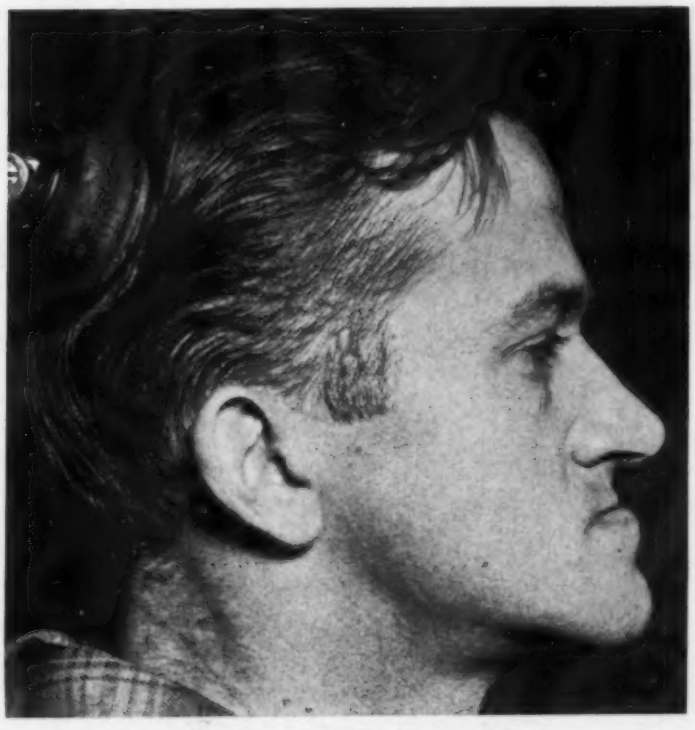


Fig. 4.—Case 2. Profile before osteotomy.



Fig. 5.—Case 2. Profile after operation. The means of securing the splint to the head cap is shown, and the wire in the mastoid region, which is inserted in a hole in the angle of the mandible, can be seen.

acrylic. It was cemented and wired to the lower teeth. This same appliance was constructed so that it fit the upper edentulous ridge while in a corrected position. A wire loop protruded from the splint and out of the mouth. Tracing was applied to the splint upward to a plaster head cap by means of the protruding wire. Wires were attached to the mandible at the angles through drill holes. They were fixed to the head cap posterior below the mastoid process. These wires

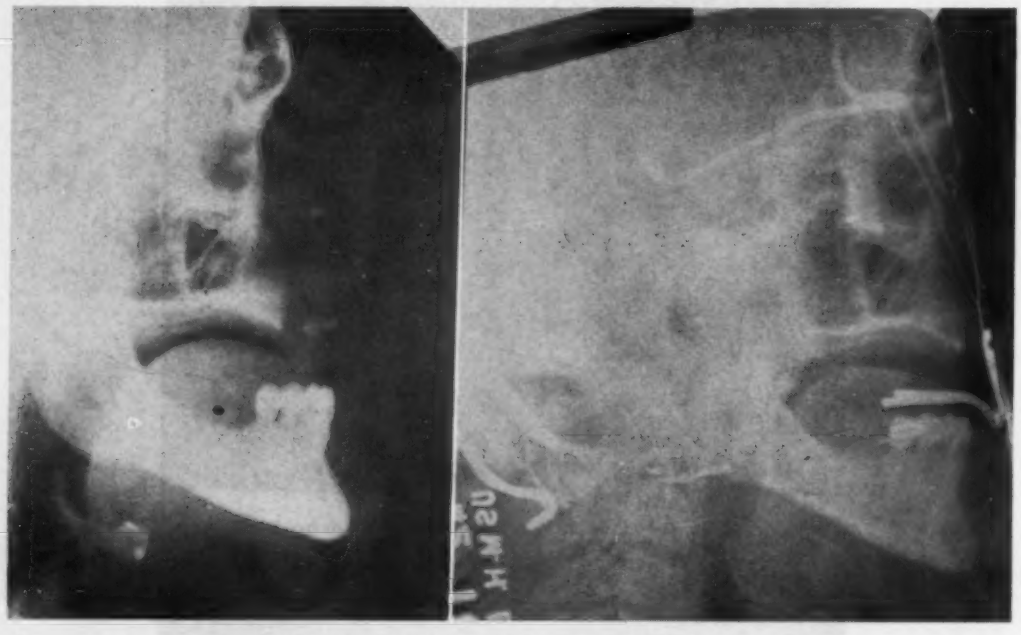


Fig. 6.—Case 2. X-ray showing relative position of mandible and maxilla before and after osteotomy.

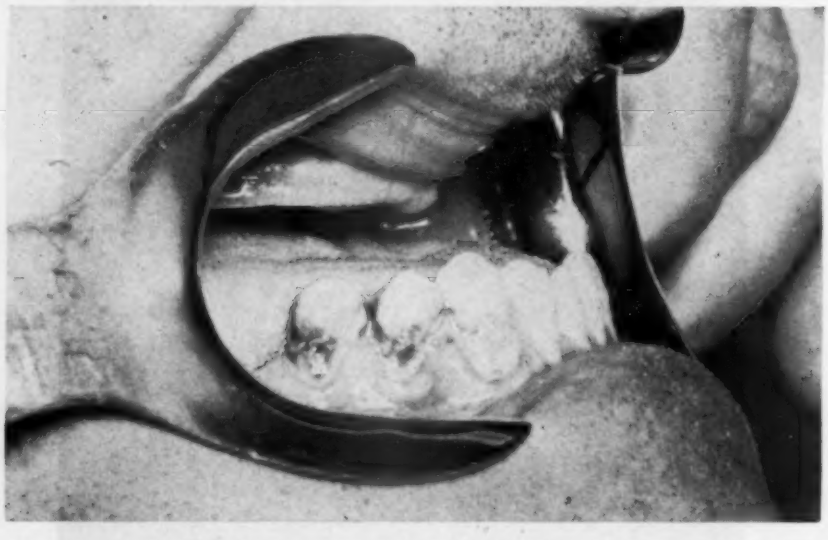


Fig. 7.—Case 2. Showing relation of upper edentulous ridge to lower natural teeth before osteotomy.

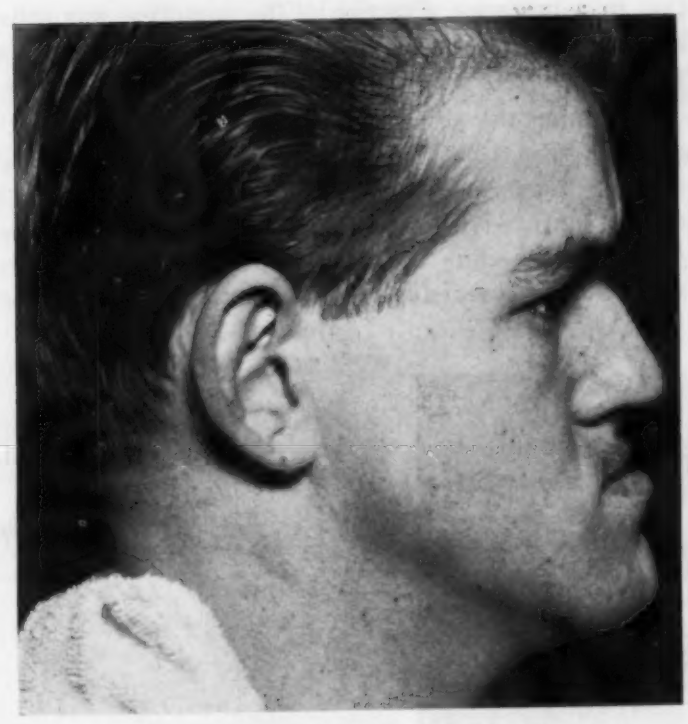


Fig. 8.—Case 3. Profile before osteotomy.



Fig. 9.—Case 3. Profile after osteotomy.

exerted traction in a posterior direction. The mandible was thus fixed in one position. Convalescence was uneventful. The splint and wires were removed two months later. Dentures were constructed, and the patient was discharged.

Several weeks later the patient returned and stated that the right side of his jaw snapped. Examination revealed a nonunion of the right side. An open reduction was performed and healing was uneventful. The patient's physical condition improved while his jaws were fixed. He gained 28 pounds and felt very well. A checkup a year postoperative was negative. His lower teeth were extracted, and a satisfactory full denture could then be constructed.



Fig. 10.

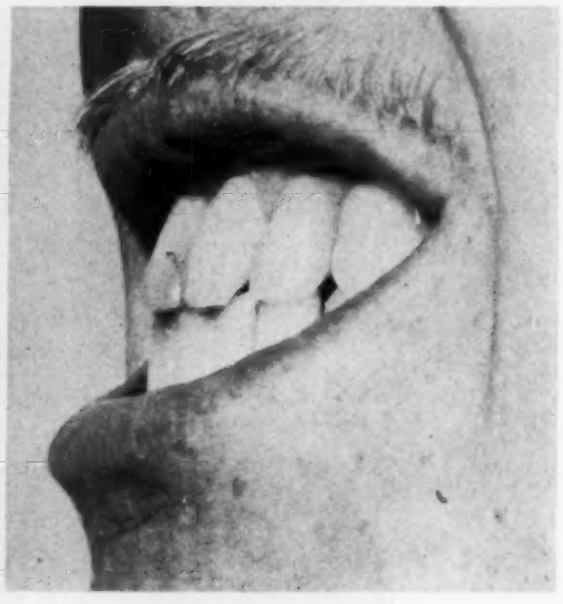


Fig. 11.

Fig. 10.—Case 3. Occlusion before osteotomy.
Fig. 11.—Case 3. Occlusion after osteotomy.

Case 3.—(Figs. 8 to 11.) F. G., a white man, aged 22 years, reported to the Dental Clinic to inquire about on operation for his protruding lower jaw. Examination showed a marked protrusion with his lower second premolars articulating with the labial surface of his upper cuspids. No other teeth occluded. A bilateral osteotomy was performed. The jaws were fixed by the eyelet method of wiring. In addition to the perpendicular interdental wires, precaution was taken to place diagonal interdental wires from the molar region above to the cuspid and premolar regions below. The wires were removed five weeks later. Convalescence was uneventful, and a very normal occlusion was obtained.

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PROSTHETIC RESTORATION OF CONGENITAL CLEFT PALATE

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THE mechanical restoration of congenital cleft palate is a problem of rehabilitation that has taxed the ingenuity of the dental profession. Every dentist who has given any thought to the reconstructive demands on his services must have realized the growing necessity for greater advancement in dental science. We are so accustomed to view the growth in knowledge from the standpoint of modern discoveries that, generally speaking, we tend to overlook the important and extensive contributions made by our predecessors, including such pioneers as Hallerius, Petronius, Paré, Fauchard, de laBarre, Snell, and Kingsley, who were skilled and successful in restoring essential structures, e.g., the orbit, nose, lip, palate, etc. They laid the groundwork on which we build.

Today among the many new substances and materials that have found use in dentistry some have become indispensable, for instance, the hydrocolloids, algenate, impression waxes, prevulcanized liquid latex, stainless steel alloys, acrylic resins, vulcanite, and methyl methyacrylate. The availability of these substances should stimulate the dental profession to further study of their possible uses in surgical maxillofacial prosthesis.

Every attempt must be made to restore normal form and function, to develop the stunted instinct of speech, to aid deglutition, articulation, to achieve a good esthetic effect, and to build up the morale of patients who are suffering from congenital defects as well as those with acquired defects. Today we are faced with increasing problems of reconstruction in acquired deformities resulting from war casualties, and we must not fail to use every facility at our command to solve them.

Those who are familiar with this reconstructive work recognize the fact that the individual, handicapped by oral and facial deformities, whether congenital or acquired through civilian or war injury, or disease, can be restored as a normal member of society. When extensive destruction has occurred and oral and plastic operations cannot restore the tissues completely, we must often resort to the mechanical restoration of lost tissue, that is, to surgical dental prosthesis. To clarify this term, surgical dental prosthetics is that branch of dentistry which aims to reconstruct congenital and acquired defects of the intraoral and extraoral structures by appliances intimately associated with surgical treatment.

CLASSIFICATION OF CLEFT PALATE

At present we are concerned with one of the most common oral defects, namely, the congenital cleft palate. This palatal deformity may be classified

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into six types (Fig. 1): I, in the azygos uvulae; II, in the uvula; III, in the soft palate; IV, in the soft and hard palates; V, in the soft and hard palates through the alveolar ridge, at the junction of the maxilla and premaxilla, accompanied by a cleft lip; and VI, in the soft and hard palates through the alveolar ridge, involving both sides of the maxilla, freeing the premaxillary bone with a double cleft lip.

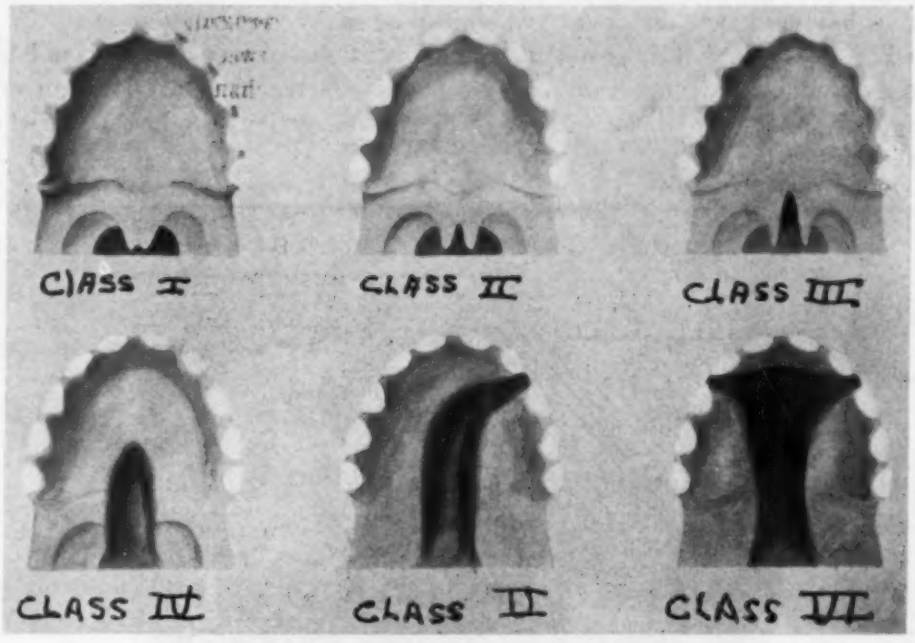


Fig. 1.—Classification of cleft palates.

ETIOLOGICAL FACTORS

Etiology.—The malformations are brought about by defective fusion or complete absence of fusion between certain structures during embryonic life. The specific cause is still unknown. Possible causes of this condition are the following: (1) Improper nourishment of the embryo. Recent experimental work points toward lack of vitamin A. (2) Heredity. (3) Mechanical influences. Pressure from the embryo's knee, tongue, or hand may tend to hold the uniting processes apart. (4) Poisonous substances circulating in the expectant mother's system, namely, drugs, alcohol, and toxins.

Anatomicophysiologic Basis.—Nature intended that we should have separate channels for the air we breathe and the food we eat. We know this is so because of the difference in the normal anatomic formation of these cavities and the histologic characteristics of these tissues. Provision is made in the nasal passages for the warming, moistening, and filtering of the air before it passes into the lungs. On the other hand, the highly sensitive mucous lining of the nasal passages is most unsuitable for contact with the different foods. In cases of cleft palate some food necessarily passes through a part of the nasal passages, producing constant irritation, which causes a chronic catarrhal condition of these tissues.

The irritation also causes the nasal tissue to secrete a viscid mucus, which contaminates the air and food in their respective passages.

The palate is one of the most important organs of speech. Its functions are concerned with (1) the formation of the tones of the voice by regulating the shape and size of the resonance chambers, and (2) closing off the nasal passages in the omission of certain sounds. This regulation being impossible, to a greater or lesser degree, in patients with cleft palate, speech is very defective, having a peculiar quality which can be easily recognized.

The soft palate then is not merely a partition between the oral and nasal passages but a very important part of the speech mechanism. It is an organ composed of many pairs of muscles which, when active, produce a variety of movements of the tissue.

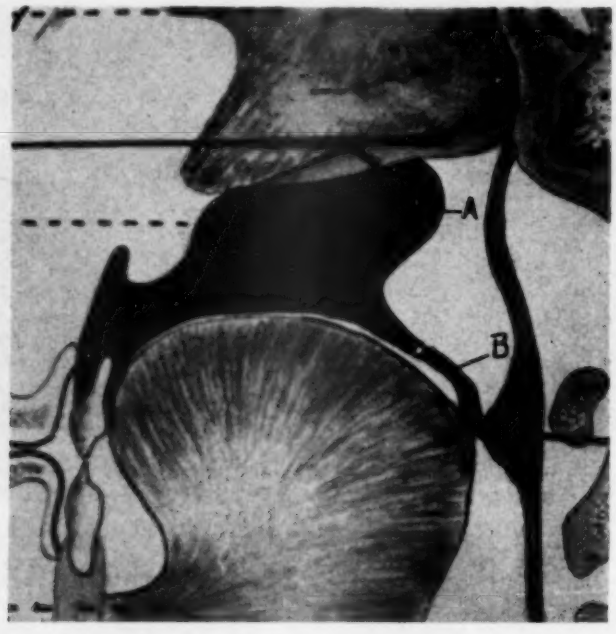


Fig. 2.—Schematic drawing. *A*, Vomer forming two separate channels of the nasal cavity. *B*, Velum connected to pinlock hinge closing off the nasopharynx cavity.

The nasal cavities with the turbinate bones, superior, middle, and inferior projections on each side, when separated by the septum, with the hard palate beneath, form six distinct resonance spaces. Posteriorly the large nasopharynx, another resonance cavity, is regulated in size and shape chiefly by the soft palate and uvula. The oral cavity, another resonator, acts with the tongue, soft palate, lips, and teeth to participate in the production of voice and speech. (Fig. 2.)

Whether surgery or prosthetics is employed, the object is to restore fully the deficient parts in such a manner that the muscles, in conjunction with the restoration, will be enabled to perform all the functions of normal speech.

TREATMENT

Many methods have been proposed and attempted by surgeons, with various degrees of success, for the improvement of speech, and all agree that the best

results are obtained when the operation is performed in early infancy. These children, however, owing to their inability to receive nourishment, are usually in no condition to undergo the operation. Consequently, the first and all-important assistance to be rendered the child with a cleft palate should be to make nourishment possible, for upon this depends its whole future.

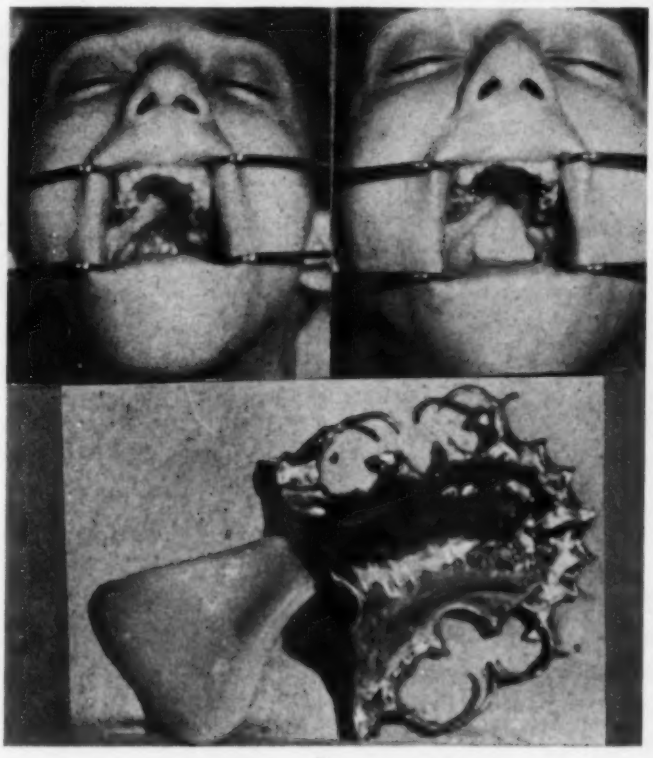


Fig. 3.—Postoperative Class III. Insufficient amount of tissues, resulting in a shortening of the uvula and destruction of the soft palate. *A*, Postoperative cleft in the soft palate. *B*, Appliance in the mouth. *C*, Retaining stress-breaking appliance with pinlock hinge and velum.

While the results are most satisfactory in many patients who have been operated upon, there are also cases in which surgery alone fails, resulting in (a) excessive postoperative tension, and (b) an insufficient amount of tissue, terminating in stretching of the soft palate and shortening of the uvula, which do not improve speech. In others, the cleft is closed, leaving a shortening of the soft palate caused by contraction, followed by scar formation. In still others, sloughing occurs and an excessive cicatrical tissue forms, preventing further surgical repair. (Fig. 3.) These defects can be greatly overcome or improved by artificial restoration of the tissues. In selected cases surgery has made prosthesis unnecessary or has greatly simplified its construction.

It is mainly on account of defective speech that the prosthodontist is consulted in cases of cleft palate. If it were possible to restore all the missing tissue surgically so that normal speech would result, the surgical procedures would be the method of choice. When surgery alone does not offer the patient the de-

sired results, then the surgeon and the prosthodontist should cooperate to the fullest extent in considering the health and comfort of the patient.

Realizing that the operation has not completely closed the opening in the palate, the oral surgeon refers the patient to the specialist in dental prosthetics, who is able to restore the missing tissues by obturators and other dental appliances. These ingenious devices not only close the opening but also stimulate the missing tissues in their proper physiologic and biologic functions. Then, with the proper speech training, the patient makes rapid strides toward a normal life.

The decision regarding the method of procedure, whether surgical or prosthetic, should be made by the surgeon and the dentist jointly.

It must be borne in mind that the number of these individuals with congenital palatal defects who can be helped by plastic surgery or artificial prosthetic restoration is greater than we realize.

Two factors are to be considered in the mechanical treatment of cleft palate, namely: (1) Improvement of tonal quality and speech to the highest degree. (2) Betterment of the health of the oral and nasal passages.

We do not say *perfect speech,* because that is hard to define. Nevertheless, in the treatment of these patients it is our duty to labor until we have at least made it possible for them to associate and converse with normal people without their defect being obvious.

Appliances of many shapes and of different substances have been devised for the closure of the cleft, but apparently with little regard for the restoration of the nasal passages, to permit normal respiration, or for the construction of the resonance chambers for the improvement of voice and speech.

In the appliances I have devised, all factors have been taken into consideration, and an attempt has been made to restore all missing tissues and their functions.

In the complete cleft where the vomer and turbinates are absent, an artificial vomer should be constructed. The vomer serves a specific physiologic purpose. In the process of inhaling, air particles of moisture collect on the vomer, and forming drops, trickle into the throat. These drops of moisture lubricate the soft tissues and thus prevent incrustation of mucus adhering to the walls of the pharynx. When this normal condition prevails, fetid breath and injury to the digestion of food do not occur.

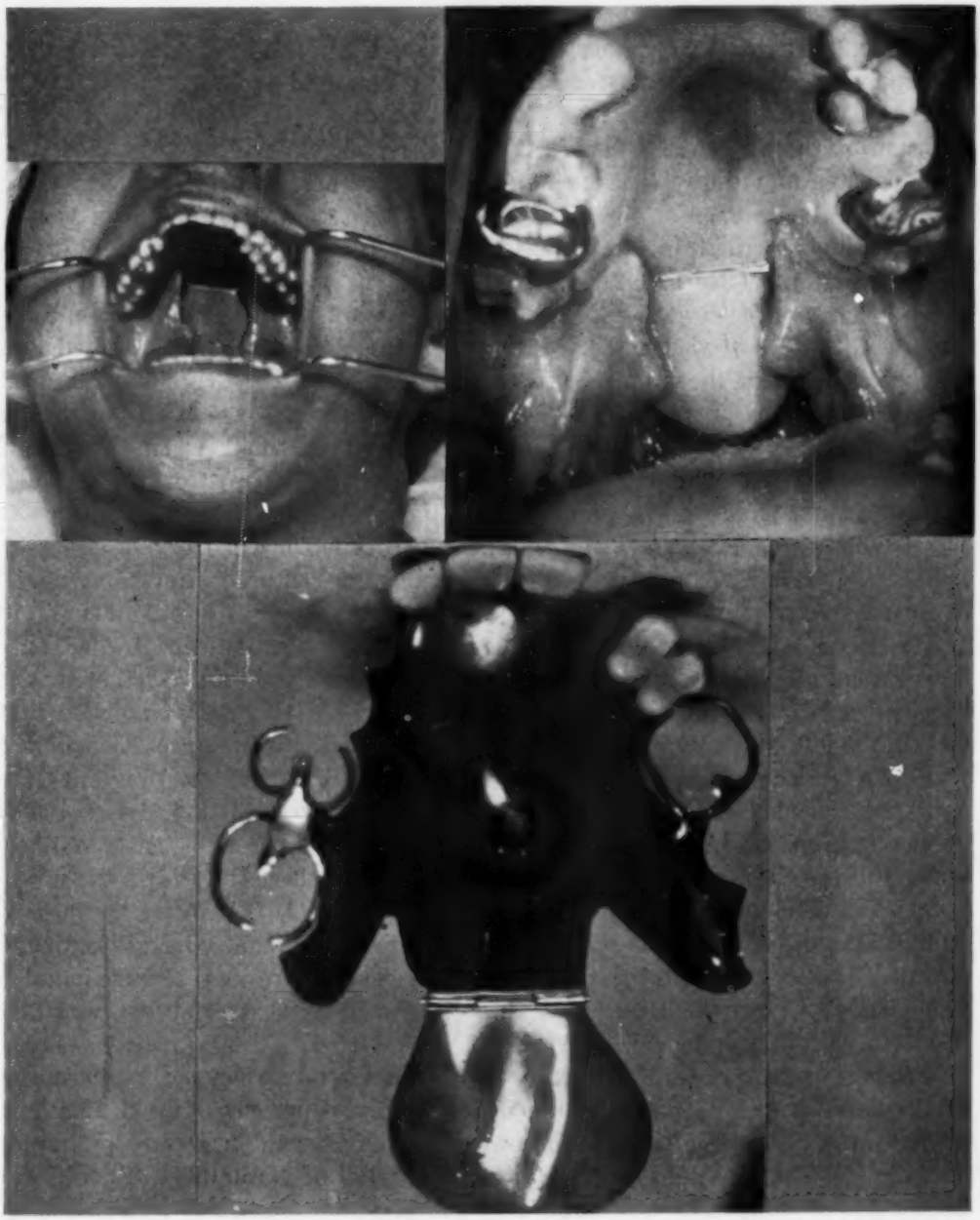
In the absence of the soft palate, an artificial velum is constructed and attached to a retaining appliance by means of a hinge, thus making it possible for the patient to pronounce those sounds in which the soft palate plays an important role (See Fig. 2, *B*).

The function of the velum palati in articulate speech is not confined to the interruption of the nasal passage; in the formation of certain sounds it must be depressed and held in firm contact with the dorsum of the tongue, and the sound must be directed and prolonged through the nasal passages. All these functions demand a palatal organ of *flexibility, mobility,* and *extent.*

The voice as it issues from the larnyx is modified in its tone and character by resonance in the buccal or nasal cavities separately and, in certain cases,

by simultaneous resonance in both cavities. Furthermore, the voice is directed or interrupted in its passage by certain organs with which it comes in contact and thus an almost endless variety of tones and combination of tones is created.

When the artificial velum is in use, the joint and the wings immediately contiguous lie close across the opening on the superior or nasal surface, hug-



A. *B.*

C.

Fig. 4.—*A*, Obturator in the mouth. *B*, The velum and the lateral wings lie close across the opening on the nasal surface, hugging the palato-pharyngeal muscles. *C*, Retaining stress-breaker appliance with pinlock hinge and velum.

ging the palati pharyngeal muscle. In construction the velum is also prolonged posteriorly until it almost approximates the muscles of the pharynx when they are in repose. Both the wings extend beyond the boundary of the cleft and rest on the surface of the soft palate, covering about ⅛ or ¼ inch of its surface. This artificial velum enables the normal palate (part of the soft palate and the pharyngeal muscle) to function in an up-and-down motion according to the requirements of articulation. (Fig. 4.)

When the organs of speech are in repose, the uvula and the soft palate rest upon the dorsum of the tongue, allowing space in the pharyngeal cavity for respiration through the nares. When these organs are in motion, a slight elevation of the palate or a contraction of the pharynx will entirely close the nasal passage and direct the voice through the mouth. Therefore, the palate becomes a valve to open or close the nares. By constructing an artificial velum, light in weight, with thin lateral wings, it will function with free mobility as a natural organ. (See Fig. 2.)

Impression Technique.—A careful examination of the mouth should be made. All x-rays of the teeth, vitality tests, and defects should be noted and kept in mind when the impression is to be taken. Any hydrocolloid material serves well for this purpose. It is desirable to obtain as accurate an impression as possible of the cleft margins and the depth of the cleft when the use of the nasal septum is contemplated. In some cases stock trays or water-cooled trays are inadequate, so they must be modified as the occasion warrants; soft impression tray metal being soldered to the tray where needed.

After a suitable tray has been selected and the required alterations made, impression material is placed in the center of the tray in sufficient bulk to insure adequate covering for all necessary anatomic detail. The loaded tray is now carefully inserted in the mouth. The heel or posterior portion of the tray is first brought into position. The placing of the tray is so managed that the impression is forced upward into the cleft and the anterior portion of the oral cavity. This prevents an excess of the material from escaping into the pharyngeal region. A little muscle trimming is done when necessary. The tray is held in the desired position until the material is sufficiently chilled; the seal is then broken carefully in the region of the tuberosity and in the mucobuccal fold, and the tray with the impression is removed. If any of the impression is broken off in the nasal cavity, it is carefully removed backward and downward with the blade of an instrument. The impression is boxed and a stone model made, which serves as the study model. A second impression is taken for a working model.

In instances of anterior clefts, or clefts not extending beyond the terminus of the hard palate, no gagging difficulties will be experienced by the patient. If the cleft extends into the velum, however, it may become necessary to employ local or topical anesthesia in the posterior palatine foramina. Should the tissues not be sufficiently desensitized by the injection, naturally it is extremely difficult to obtain an accurate impression. Another method to prevent gagging is to allow a lump of sugar saturated with a drop of spirits of camphor to dissolve slowly in the mouth.

DESIGNING AND CONSTRUCTION

Designing.—Construction is planned on the study model. Abutment teeth, the type of arch, the width of the cleft, and the length of the remaining hard palate are taken into consideration.

Working Model.—In the event of a complete cleft palate with a large opening into the nasal cavity, the procedure may be divided into two steps:

1. *Impression of the Nasal Portion.*—A piece of soft impression-tray metal is burnished over the cleft on the study model. This is perforated throughout with a sharp-pointed instrument or burr so that the roughened portion shall be situated palatally or nasally. This tray is filled with a generous bulk of hydrocolloid and gently lifted into place, where it is allowed to cool by spraying cold water into the mouth.

2. *Impression of the Palate.*—While the nasal impression is in place, a hydrocolloid impression of the mouth is taken. The oral and nasal impressions are carefully removed. The nasal impression is then placed in the proper position and a model is poured.

Construction of Obturator.—The cleft is filled with hard wax and made to conform to the remaining lateral plates of the palate, so that a normal contour is given to the roof of the mouth. The design previously made on the study model is now carried out. A baseplate is constructed, and a bite is obtained against the lower teeth. Careful consideration must be given in constructing the proper clasps. Where the teeth are closely approximated, the Jackson crib clasp is indicated; where the teeth are separated the Bonwill or Gillett or Roache clasp is ideal. In most cases two clasps are generally sufficient to retain the denture.

Clasps are bent into shape, teeth are ground into occlusion, and waxed into position. If the "try-in" is satisfactory, we are ready to construct the vomer attachment. First, the pinlock-hinge attachment must be accurately positioned. This attachment is constructed in two parts. One-half of the attachment is waxed in the palatal portion of the denture, or obturator. This must be accurately placed in the posterior extremity of the palatal portion, so that the other half portion fits in the exact center of the pharyngeal opening. When this section of the obturator is satisfactorily adjusted, it may be made in vulcanite, in light metal alloys, or, preferably, in acrylic resin.

The vomer is made of a double thickness of baseplate wax; it is formed into the shape of a rectangle and is seared to the nasal aspect of the trial plate. While it is in the soft stage, it is placed in the mouth. It is then removed and the excess is trimmed off and a border of carding wax is traced on the periphery. Black carding wax is used or a soft wax may be made by melting equal parts of pure beeswax and paraffin. The obturator is again inserted in the mouth after the carding wax has been traced, and then removed. This is done as often as necessary to make it conform to the remnant of the remaining vomer.

After the above section of the denture is completed in vulcanite, acrylic resin, or metal, we are ready to work on the velum. Since the attachment or

part of the pinlock hinge has already been inbedded in the denture, the other half of the hinge is connected with the fixed hinge in the denture, so that it hangs posteriorly in the nasopharynx. A triangular piece of baseplate wax of sufficient width is added to the loop so that it contacts the pharyngeal wall

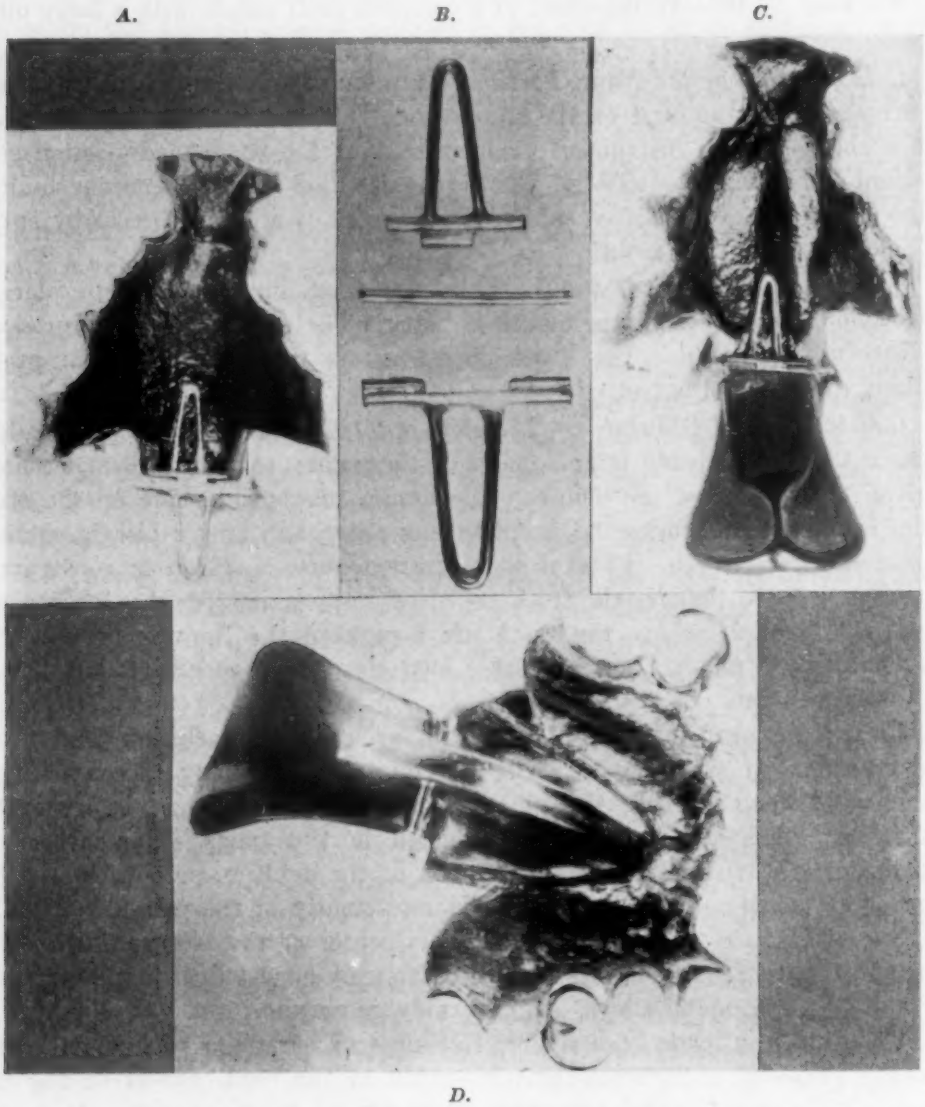


Fig. 5.—Construction of a pinlock-hinge retaining appliance with vomer and velum. *A*, Part of the hinge is imbedded in the posterior tongue of the trial retaining appliance. *B*, Iridium-platinum pinlock hinge. *C*, Velum connected. *D*, Vomer attached to the nasal aspect conformed to the remaining remnant in the nasal cavity.

and extends to passavant's pad at the superior constrictor muscle. The patient is asked to swallow, turn his head sidewise, backwards, and forward. (Figs. 5 and 8.)

The denture is removed, a careful examination is made of the velum, slightly more than the excess is removed, and it is then traced with carding

wax. The described exercise is repeated several times until the velum extends to Passavant's pad. When the patient swallows, the palatopharyngeal muscles should grasp the velum to shut off the nasopharyngeal cavity. This forms a seal, preventing fluid or food from passing into the nasal cavity.

The patient is told to exercise by pronouncing the guttural letters such as *k*, *c*, *a*, etc. The obturator with the velum is again removed from the mouth, and either the excess is removed, or soft wax is traced where there is deficiency. When the patient expresses comfort, the appliance is ready for processing.

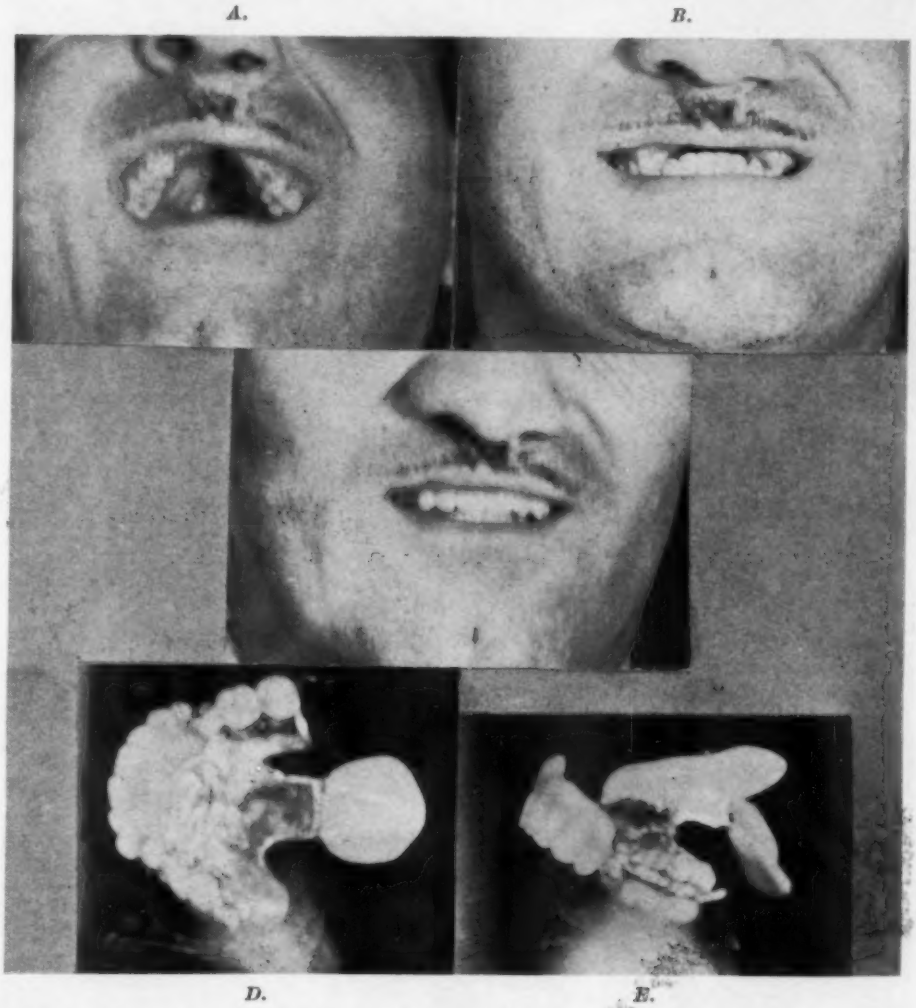


Fig. 6.—Class VI cleft palate. *A*, Class VI cleft palate. *B*, Premaxillary and six anterior teeth missing. *C*, Bite opened 6 mm. with acrylic onlay molar cusps. *D*, Palatal view. *E*, Nasal view.

When the obturator is completed, it is inserted in the mouth and any necessary corrections are made. The patient may make several visits for minor adjustments and is then taught to practice phonetic sounds, e. g., the vowels as *a* in ate, at, ah, followed by the sounds of all the explosives, e. g., *g* as in give, *k* as in king, *q* as in quite, and so forth. The object of this exercise

is to strengthen and develop the pharyngeal and palatal musculatures, so that they will firmly and involuntarily close around the artificial velum, completely preventing the vocalized air from escaping into the nose.

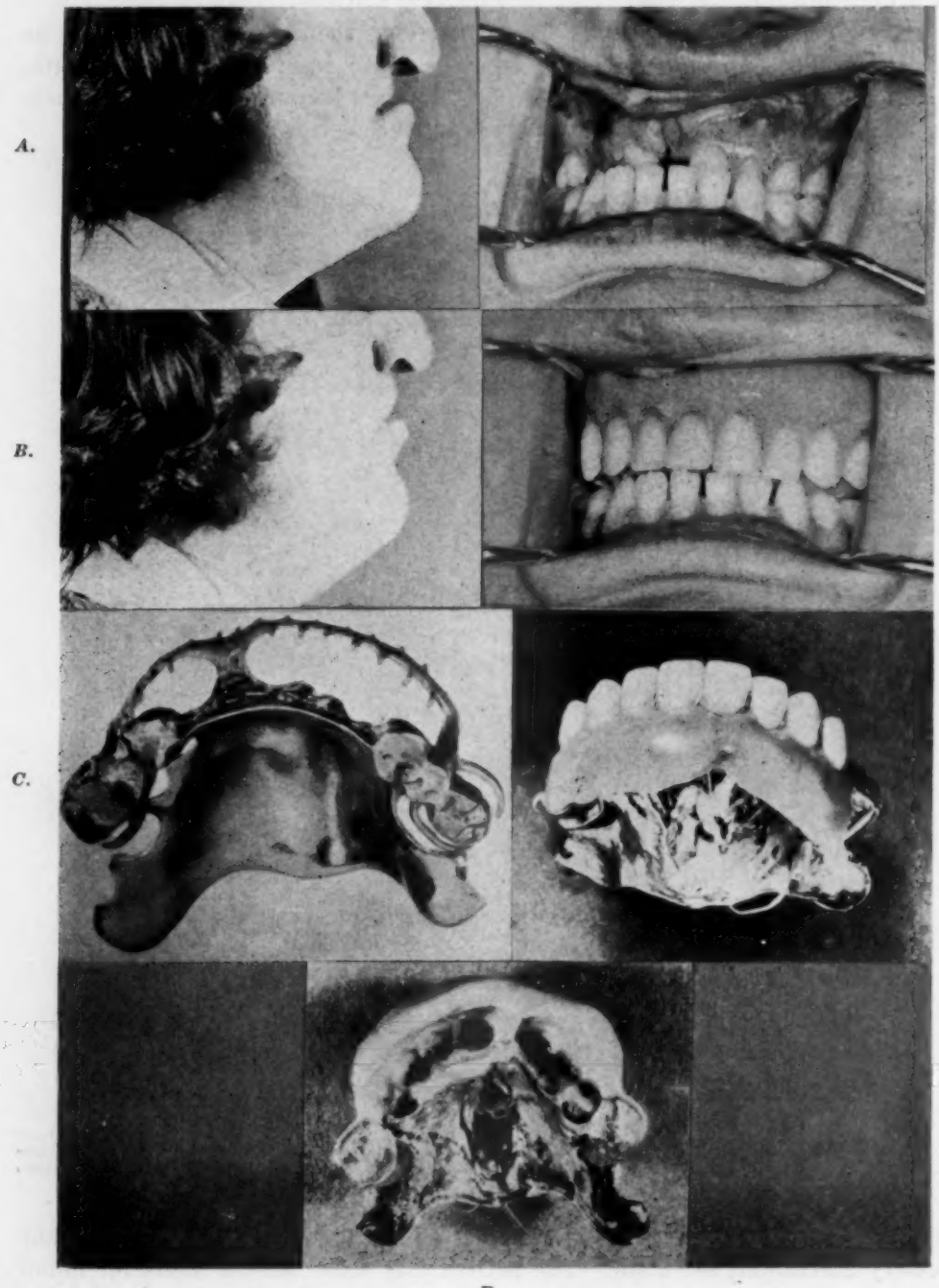


Fig. 7.—Class V cleft palate. *A*, Before. *B*, After. *C*, Skeletal retaining appliance, anterior frame for acrylic retention. *D*, Palatal view. *E*, Anterior view.

Improved speech and articulation depend greatly upon the muscles of the patient to function properly, and upon the desire and will to continue his effort until a measure of success is attained. It is essential that speech lessons be given by a teacher who really understands the speech problems of a patient with cleft palate.

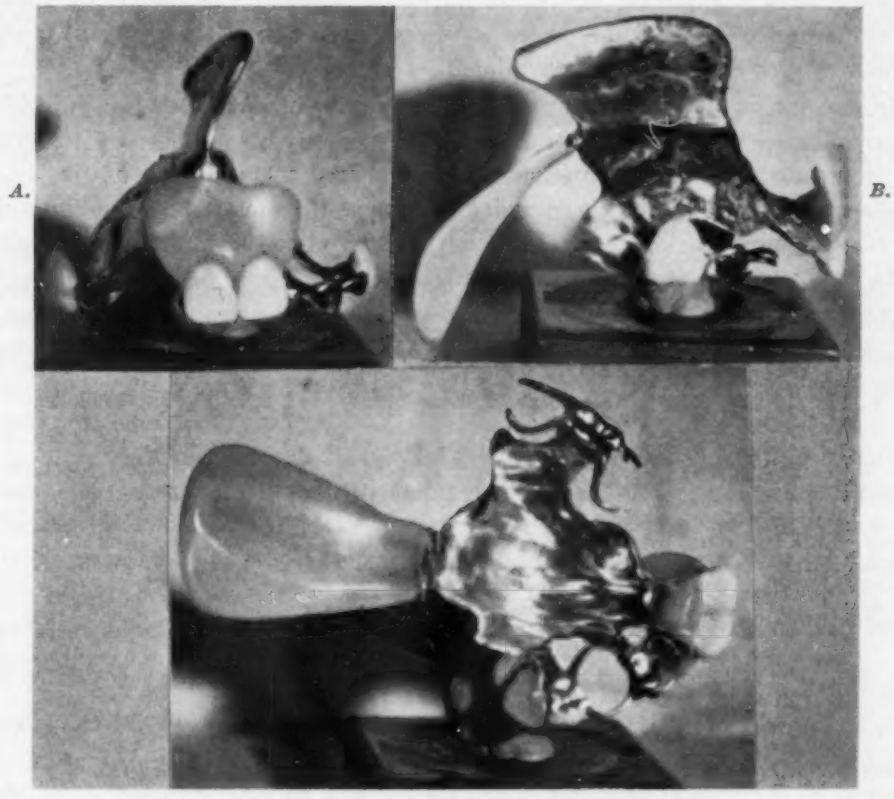


Fig. 8.—Obturator appliance for Class V cleft palate. *A*, Front view premaxillary. *B*, Palatal view. *C*, Vomer on the nasal aspect, flexible velum with pinlock hinge.

PSYCHOLOGICAL EFFECTS

Any of the facial and oral deformities accompanying cleft palate may have grave psychological effects. When these unfortunate persons so afflicted attempt to speak, they become conscious of their pronounced defect, which has a depressing influence upon their mental and physical development and well-being. Consequently, the child, male or female, and particularly the latter, born with cleft palate, starts life with a severe social handicap. Correction of the deformity and prevention of the psychic inferiority that comes with the defect in speech and appearance convert a eugenic liability into a eugenic asset. (Figs. 6 and 7.)

The probability that the child with cleft lip or cleft palate will become a normal person is excellent, provided the parents will do their part intelligently and faithfully. This requires a program which involves strict supervision for

several years, first by the surgeon, then by the dentist, the nurse, the speech expert, the family doctor, and the family itself. This program must be begun on the day the child is born. The first problem is that of feeding and stimulating the natural reflexes of sucking and swallowing. Feeding will be easier after the lip has been surgically repaired.

Since such a program of rehabilitation is beyond the means of families with moderate or low income, there is urgent need for an institution or clinic in which proper care would be available.

In conclusion, I wish to point out that the rehabilitation of the individuals who are afflicted with cleft palate is a purpose worthy of our best effort. Think what it means to them. The correction of such defects, as far as lies in our inventive power, alleviates their social isolation and makes possible the fulfillment of their normal desire to mingle with their fellows without embarrassment and to enjoy what life holds in human contact.

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ORAL DEFECTS IN THEIR RELATION TO DEFECTIVE SPEECH

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BEFORE attempting to correct speech defects by training, it is important to make sure that the speech mechanism is not defective. We do not expect to get full power from the engine of our automobiles if one or more of the cylinders are out of commission. Nor can we expect to find perfect speech in an individual who cannot use the full power of the speech mechanism, because of some mechanical defect in it.

We are all familiar with the snuffling hollow sound which is characteristic of cleft palate cases; but to this more obviously recognizable deformity may be added deformed dental arches (teeth not in their normal arrangement and alignment), paralysis of the soft palate, alteration of the cavity by growths, and faulty artificial dentures. We may also have teeth so abnormal in position that their functions are greatly interfered with, although the deformity may be scarcely noticeable.

Correcting these mechanical defects wherever possible should come first before speech training is attempted. Sometimes the adjustment of the malformation is sufficient and the patient can speak with ease. In other cases, training is necessary because the patient has established faulty habits of speech which still persist, even after the cause has been removed. For example, a lisp caused by a missing front tooth may continue after a false tooth has been inserted, simply because the individual has formed the habit of lisping.

Training starts after the mechanical defect, the original cause of the difficulty, has been removed. Where speech defects have arisen because of oral defects, training should be entrusted to a specialist who has a thorough understanding of oral defects in their relation to defective speech and a complete knowledge of the complex vocal mechanism.

If the vocal cords alone were responsible for speech, there would be few problems, but actually the vocal cords by themselves are capable of nothing more than a feeble and disagreeable sound. Coming to the assistance of the vocal cords are the lungs, which, acting as bellows, furnish the current of air to make the cords vibrate. Above the vocal cords are the laryngeal, pharyngeal, oral, and nasal cavities, all of which are resonance chambers. These resonance chambers by their muscular arrangements are capable of being changed in shape and size, and can thus be attuned to the pitch of the sound produced by the vibration of the vocal cords. It is the quickness and precision with which the different movements are executed in these cavities which makes what is called a trained voice. A voice seemingly weak in volume and poor in quality can be improved to a great extent by such training.

In addition to the lungs and the resonance chambers, in the oral cavity there are the tongue, teeth, roof of the mouth, the soft and hard palate, lips, and cheeks, all concerned in the production of speech. Of these, the functions of the palate are the most important. Not only is the palate essential in the enuncia-

tion of nearly all the elements of speech, but owing to its direct attachment to the larynx, it is also an important factor in the production of voice. The vowel sounds may be articulated when the palate is defective, but their resonance is so much impaired that they are scarcely recognizable and their pitch cannot be changed with any degree of accuracy as in the case of the cleft palate. It is in the articulation of consonant sounds, however, that the palate is especially essential.

The consonant sounds are made by impeding the moving column of breath at certain points above the larynx. The points at which the impediment takes place have been called the stop positions. These have been divided into the anterior, the middle, and the posterior stop positions. The anterior one is formed by the lips, by the lower lips and teeth, and by the tip of the tongue; the middle one by the tongue and hard palate, and the posterior one by the dorsum of the tongue and the soft palate.

The function of the palate in articulation of consonant sounds, therefore, is twofold. In all those sounds in which it does not assist in the formation of stop positions, it serves as an obturator between the nose and the pharynx, completing the partition between these two cavities and compelling the outgoing breath to pass through the particular stop position required for the sound. For example, in the articulation of labials (*p, b, m, wh,* and *w*) labiodentals (*f* and *v*), and labiolinguals (*th,* voiced and unvoiced), the sounding breath must pass through the anterior stop position, and the palate serves to diverge it in this direction and to prevent it from passing through the nostrils. In a similar manner, when the hard palate is intact and the middle stop position is used, as in the articulation of linguopalatals, the sounding breath must pass through this constricted aperture and the function of the palate is to prevent it from passing upward through the nostrils.

In the use of the posterior stop position, which is formed by the junction of the velum palate and the dorsum of the tongue, the soft palate serves a double purpose. Its free border rises against the posterior pharyngeal wall, closing the avenue to the nostrils, and its anterior surface, acting in conjunction with the tongue, forms the stop position for the sound.

The soft palate has a wide range of movements of great importance to the perfection of speech. Its function in vocalization is to assist in controlling the action of the vocal cords and regulating the size and shape of certain important resonance chambers, and its function in articulation is to shut off the nasal from the oral cavity during emission of explosive and so-called posterior linguopalatal sounds.

In the case of the cleft palate, since a part of the speech mechanism is missing, it is necessary to resort to either surgical or mechanical means, before any improvement in speech can be expected. After the operation for closure of the palate has been performed, treatment of these difficult cases should be begun. Since there is apt to be a weakness and lack of development in the upper air passages and in the nasopharynx, interfering with the timber of the voice and with the articulation of certain letters and syllables, it is essential that corrective exercises be given to strengthen and stimulate the upper air passages and to help establish a more healthy state in the nasal and pharyngeal passages.

Great improvement may be brought about by breathing exercises devised to reach these parts by way of the nose, because breath taken only through the nose exerts a favorable stimulus on these enfeebled upper airways and helps bring about a more healthy and normal state in the nasal and pharyngeal passages.

Another characteristic of cleft palate speech is the disagreeable nasality of tone, which is caused by the imperfect and immobile soft palate. The operation of uniting the fissure, which usually extends from the hard palate through the soft palate completely dividing it, causes some amount of thickening and loss of elasticity. Even where there is no thickening, the soft palate may be immobile because of the loss of nasal breathing.

Great progress has been made in the surgical treatment of cleft palate, thus making it possible through training for the patient more nearly to approximate normal speech. In some cases when the operation is made in infancy and training begins early, no deviation from normal speech can be noticed.

If, in closing the upper jaws, a space is left where unerupted or missing teeth should have come, it is impossible to get the true sound of certain letters as *s*, *c* (soft), *sh*, and *z*, unless artificial teeth are inserted. Sometimes the missing tooth erupts through the middle of the hard palate and unless it is extracted by a dental surgeon, it not only interferes with the patient's speech, but also makes the tongue rough and sore.

In the absence of the soft palate, an artificial velum is constructed and attached to a retaining appliance, thus making it possible for the patient to pronounce those sounds in which the soft palate plays an important part. This arrangement is good for nearly all letters except hard *g*, the closure for that letter taking place lower in the throat than the artificial velum can reach. If there is only a portion of the soft palate, or if it is slightly paretic, exercises must be given to overcome the condition.

If there has been a cleft lip as well as a cleft palate, the lip is sometimes shortened by the operation for closing it, or the muscles are stiffened. In this condition, it is difficult for the patient to pronounce the letters *b*, *p*, *m*, *w*, and *y*.

In cleft palate cases, following surgery, the soft palate is almost always inelastic and immobile. Sometimes there is very little left of it after the operation, and it is entirely too short to shut off the nose from the throat. In other cases, the edges are very thick, and occasionally the closing seam has destroyed the power of movement, but a great deal can be done by educational measures to restore the function through exercises designed to bring about the power of using the various muscles involved in a manner as nearly as possible approaching that which is natural to the normal. Nature can be very adaptable if given a chance.

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ORAL SURGERY CASE REPORTS

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CASE 1. BILATERAL FRACTURE OF MANDIBLE

THE patient, chief commissary steward, aged 50 years, was struck by bomb fragments from a Kamiekaze attack on Dec. 7, 1944, in the Philippine area while engaged in organized action with the enemy. He regained consciousness in the water after several hours. His injuries were multiple, the most serious involving his jaw, his right elbow, and head.

On December 12 he was taken to a U. S. Army hospital in New Guinea where screws were placed in the mandible and a cast applied over the area.

On December 24 he was removed to a hospital ship where a Roger Anderson appliance was applied in an effort to reduce the fractures.

On Jan. 10, 1945, external fixation was considered unsuccessful and the pins were removed. The reasons as far as we could ascertain were: (1) the frailty of the osseous structures, (2) inability to keep the pins firmly imbedded in bone, (3) muscle spasm and traction, (4) bone degeneration, sloughing, and suppuration which indicated possible osteomyelitis. He was transported to San Francisco, and arrived at this hospital on February 9.

Oral examination revealed the mouth to be edentulous. The patient had worn full maxillary and full mandibular dentures for ten years; these were lost at the time of injury. The mandible was delicate and small. There was an ununited fracture on the right side at about the area of the first molar. There were two main ununited fractures of the left side; one anteriorly, in the area of the mental foramen and one posteriorly, at the angle of the mandible (Figs. 1*A* and 1*B*). External scars which exhibited no drainage were noted. Intraorally the mucosa was completely healed and uninterrupted, with a marked retrusion and elevation of the anterior segment. The chin was greatly depressed by traction of the muscles of the floor of the mouth; the patient was unable to close his mouth. Moderate mobility of the segments and crepitus were felt, indicating that the fractures were ununited but were probably held by partial fibrosis.

The patient was disfigured and was considered a dental cripple inasmuch as it would be impossible to make prostheses in the existing state.

General Considerations.—(1) *Pin fixation:* We agree with Erich and Austin[1] that in edentulous jaws with multiple fractures the most satisfactory means of immobilization is pin fixation. However, prior pin fixation in this

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The opinions or assertations contained herein are the private ones of the writer and not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

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Fig. 1*A*.—Case 1. Fractures of mandible at time of admittance on Feb. 13, 1945. Downward displacement of anterior segment with telescoping into rami. Metallic foreign bodies in right mandible imbedded in soft tissues.



Fig. 1*B*.—Lateral view of fractures.

case had yielded unsatisfactory results. The contraindications for repeating reduction by pin fixation were the delicacy of jaw structure and the long interval of time since injury had occurred.

2. *Open reduction:* It is also generally accepted that open operations for reduction or fixation of fractures of the jaws too often end in osteomyelitis and necrosis of bone or nonunion.



Fig. 2.—Case 1. Rigid plaster head cap with anterior arch bar and posterior extension bars; swivel attachment to regulate direction of tension and wing nuts to control degree of tension. (Official U. S. Navy Photograph.)

3. *Surgical prosthesis:* Erich and Austin state: "If osteomyelitis is to be avoided we believe that open operations are never to be undertaken. One should rely on and should have enough ingenuity to construct intraoral or external appliances which do not require surgical exposure of the line of fracture but which supply adequate immobilization of the fragments of the jaw."

As a result of experiences in this war, it may be frankly stated here that the use of the sulfonamides and penicillin therapy has modified this clinical assumption. Nevertheless, we selected a conservative approach and the following treatment was carried on:

First Operation.—On February 17 the patient received preoperatively morphine sulfate, $\frac{1}{6}$ grain, and atropine sulfate, $\frac{1}{150}$ grain. Under pentothal sodium intravenous general anesthesia, and according to the Ivy and Curtis[2] technique small incisions about $\frac{1}{2}$ inch long were made through the skin to the lower border of the mandible, at three sites: the symphysis, the right and the left angles of the ramus. By blunt dissection, tissues and muscle fibers

were pushed aside and retracted, and the bone was exposed. Holes were made through the mandible by using a surgical hand drill. Stainless steel 25 gauge wire in double strands was passed through and twisted one turn and allowed to extend externally. The wounds were closed by subcuticular sutures and the skin incision by fine dermal sutures, without insertion of a drain.

On February 23 a plaster head cap (Fig. 2) was constructed, imbedding in the plaster a rigid anterior arch bar and two posterior bars to which the stainless steel wires from the jaw were connected (Fig. 3).

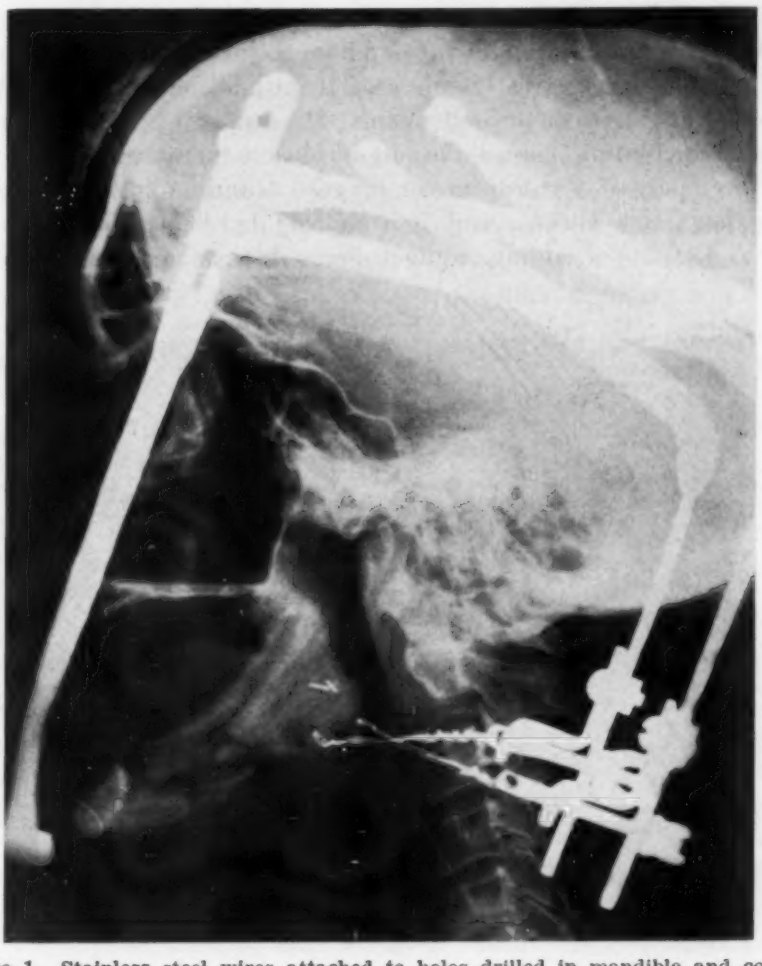


Fig. 3.—Case 1. Stainless steel wires attached to holes drilled in mandible and connected to attachments in plaster head cap. Note extent of reduction in one week.

This appliance functioned efficiently, as will be seen in Fig. 2. The two short bars posteriorly were rigid and their function was to stabilize the mandible. The anterior face-bow had a moderate degree of elasticity and helped to maintain a gentle continuous traction. The swivel attachment made it possible to change the direction of traction if necessary. The three wing nuts varied the length of wires and controlled the tension to the extent which the patient was able to tolerate. Most of the reduction was completed in the first ten days, and thereafter the appliance served to immobilize the fractures.

During the six-week period in which patient wore this appliance with complete fixation of the mandible, his chief complaint was pressure sores developing under the head cap through indirect pressure. High caloric, liquid, and very soft diet furnished nourishment without much difficulty in deglutition.

Second Operation.—On March 10, again under pentothal sodium general anesthesia, a second operation was performed. Circumferential wiring was inserted in four places over a previously constructed acrylic splint. This appliance was occluded with a full maxillary splint.

Technique of Circumferential Wiring.—In the method described by Fry, Shepherd, McLeod, and Parfitt,[3] a lower acrylic splint is fixed across the fracture by wires passed around the mandible in the soft tissues. The immobilization of the fracture is greatly enhanced if, in addition to the circumferential wires, the lower jaw is stabilized against the upper by a chin bandage and upper plate articulating against the bite blocks on the lower. "In other words it is the lower part of a vulcanite (or acrylic) Gunning splint that is wired to the lower jaw; this lower splint is stabilized by being articulated *but not fastened* to the upper splint, contact being maintained by an elastic chin bandage."



Fig. 4.—Case 1. Acrylic splints in mouth and circumferential wiring. Spaces provided for feeding. Splints immobilized by skull sling.

Fig. 4 illustrates the splints used for this patient.

The circumferential wires were placed by the following modified technique:

1. The position of the grooves in the lower splint were marked in the mucous membrane.

2. At the lower border of one of the fractures a small incision was made through the skin and a curved 15 gauge hypodermic needle passed through this incision close to the bone on the lingual side, until the mucous membrane of the mouth was pierced. A 25 gauge stainless wire was passed through the lumen of the needle and the needle was withdrawn, leaving the wire in place.

3. The needle was inserted again in the buccal sulcus and passed *downward* from the mouth in a similar manner close to the bone, this time on the labial side until its point emerged at the original skin incision. The other end of the wire was then passed up through the lumen of the needle and the needle was withdrawn into the mouth. The wire was passed around the bone with the ends emerging in the mouth.

4. Both ends of the wire were firmly grasped and with a sawing movement the wire was made to cut through the soft tissues so that it came to lie against the lower border of the mandible.

5. The ends of the wire were now twisted over the splint so that the bone fragments were drawn up snugly in contact with the splint.

The day before the operation the plaster head cap had been discarded, but the traction wires placed on February 23, were still retained, protruding from the skin. After a few more days, when it was felt that the splint would perform its intended function of fixation, they were removed.

With the second insertion of the needle downward through the buccal mucosa on the left posterior region, profuse hemorrhage was detected from the lingual aspect around the wire. Puncture of the lingual artery was suspected, probably due to scarring and distortion of the tissues. The hemorrhage was not controlled until digital pressure was applied externally on the mandibular notch. Later, tightening of the wire over the splint produced complete hemostasis, probably by compressing the external facial artery. In the meantime, the patient had lost an excessive amount of blood and two units of plasma were given immediately in the operating room. The immediate postoperative condition of the patient was satisfactory but recovery was not uneventful. The patient ran a moderately febrile temperature. The blood pressure was moderately high and the red blood count was low.

On March 16 the patient developed multiple cutaneous abscesses, starting at the sites of incision and wire insertion and spreading under the chin, with copious suppuration and free drainage. Culture of the abscesses revealed *Staphylococcus albus hemolyticus.*

With the first signs of infection, intravenous penicillin therapy was started under the supervision of the Ward Medical Officer, for a total period of fifteen days. On the first six days the daily penicillin dosage was 150,000 units. Medication was supplemented orally with daily dosages of sulfadiazine and sodium bicarbonate, 2 Gm. each, and ferrous sulfate, 2 Gm. Discontinuing sulfadiazine, the remainder of the treatment consisted of penicillin intravenously, 200,000 units daily. The immediate results were a marked improvement and a tendency to localization.

On March 23 the red blood count was 3,358,000, and the hemoglobin 10 Gm. On March 24, 500 c.c. of citrated whole blood were given intravenously, following which improvement was very rapid.

Other treatment included therapeutic doses of vitamin B complex, vitamins A and D, and calcium hypophosphite. The patient's diet was fortified by prescribing eggnogs and forced fluids to accelerate healing and reduce convalescence time.

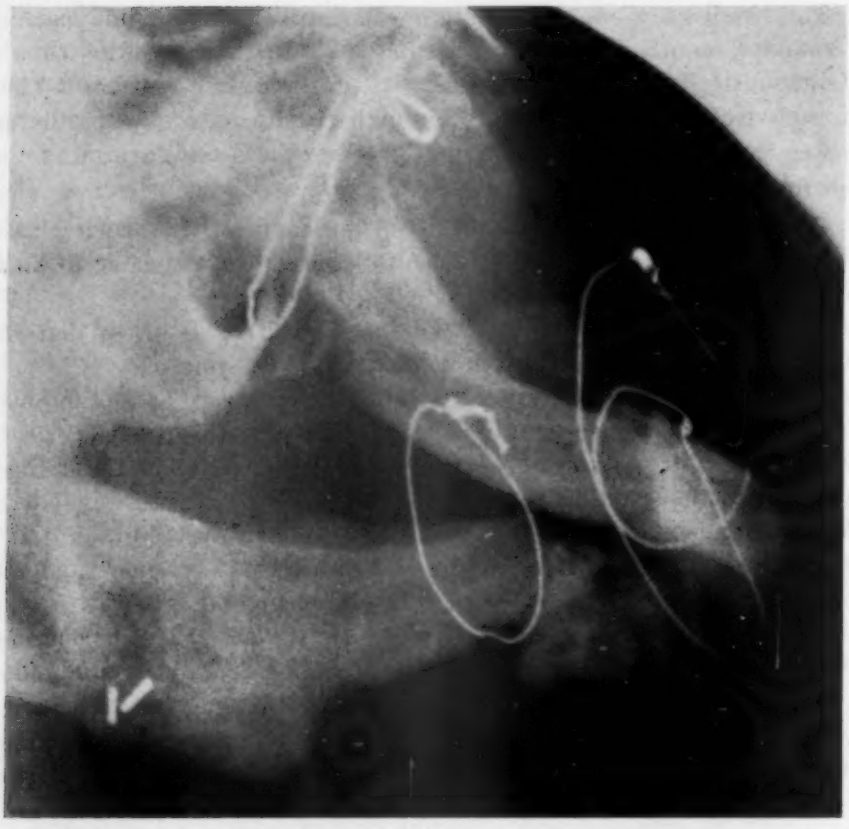


Fig. 5.—Case 1. April 30, 1945. Showing union of fragments by bridging. Note that inferior border of left mandible is scalloped.

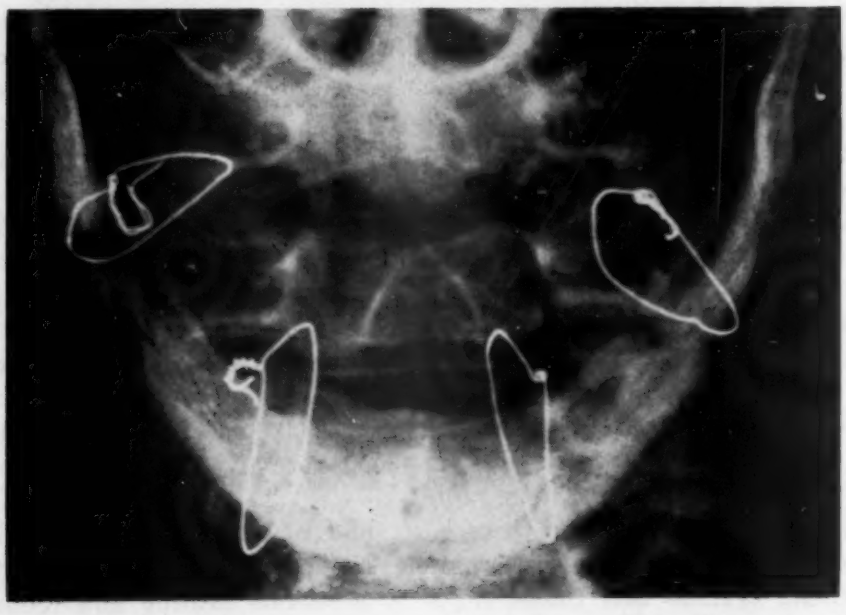


Fig. 6.—Case 1. Circumferential wires over splint holding fragments in position.

In spite of the development of cutaneous infection, the circumferential wires were well tolerated and were left in place for four weeks. Following x-ray verification of bone union and callous (Figs. 5 and 6), the wires were removed without anesthesia, by cutting one end short. The patient presented a functional mandible on which prostheses could be constructed.

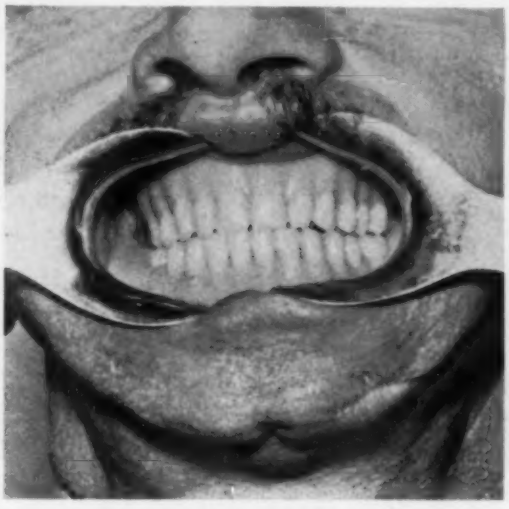
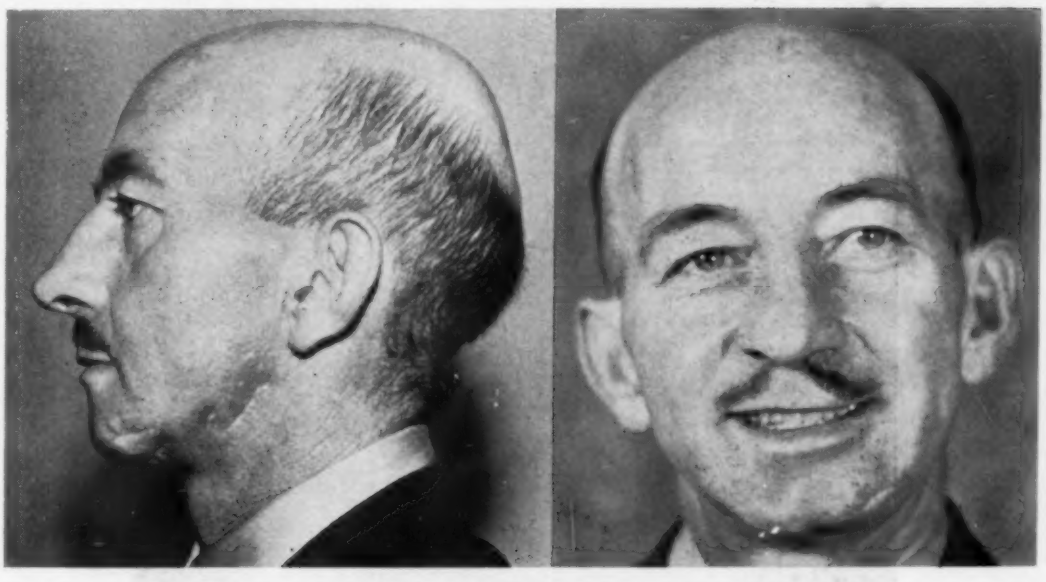


Fig. 7.—Case 1. Dentures inserted.



Figs. 8 and 9.—Case 1. Normal profile and front view of patient with dentures.

Full maxillary and mandibular dentures were inserted on April 14 by Lt. Comdr. G. B. Throupe (DC), USNR, at the U. S. Naval Center, Prosthodontia Department (Figs. 7, 8, and 9). On June 22 following his return from convalescent leave, the patient was examined and the following observations noted (Figs. 10 and 11):

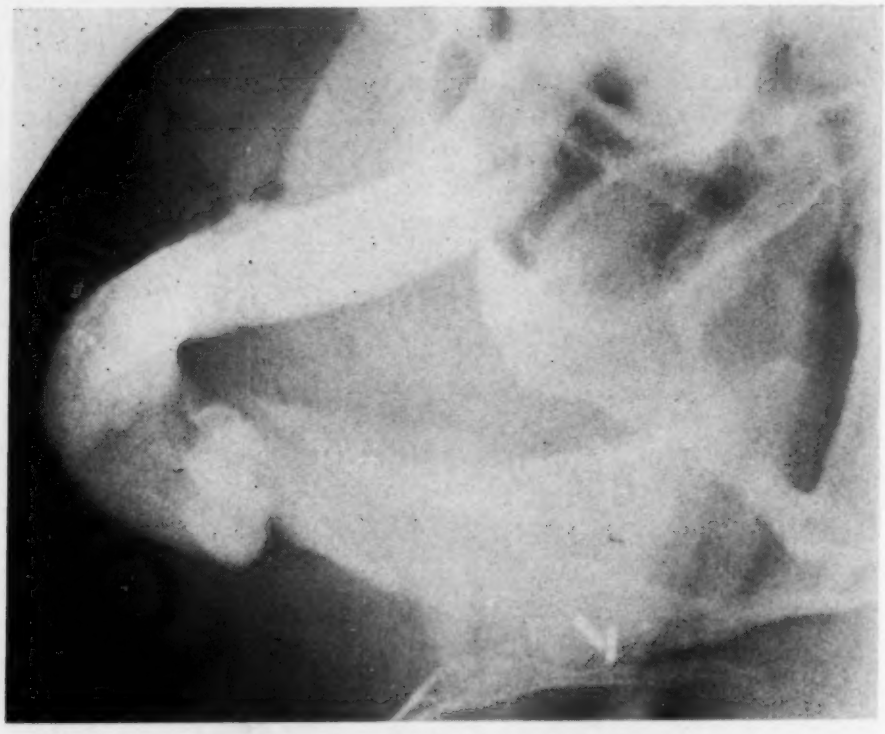


Fig. 10.—Case 1. May 19, 1945.—Complete union of fractures. Patient has worn dentures for six weeks.



Fig. 11.—Case 1. Position and union of fractures.

1. Anesthesia of chin.
2. Contour of alveolar ridge broad posteriorly.
3. Oral mucosa normal.
4. Patient could open mouth wide but lateral excursions of the mandible were limited.
5. External scars were present on skin.
6. Patient chewed well, ate moderately hard food, and was satisfied with his dentures from functional and esthetic viewpoints.
7. No evidence of infection was present. Fixation of fragments was considered complete, with satisfactory approximation for normal function of the mandible, broad ridges affording good foundation for prostheses. Prognosis for nerve regeneration and more efficient masticatory function was considered to be favorable.

CASE 2. GUNSHOT WOUNDS OF JAW

The patient, private first class, U.S.M.C., aged 27 years, in March, 1945, was a casualty on Iwo Jima, suffering extensive injuries to the maxilla, the loss of several teeth, and disfigurement of the face. A bullet had entered through the left cheek, passing through the antrum, destroying the alveolar process, and had emerged through the right cheek.

When seen at this hospital on April 12 the following observations were made:

1. Avulsion of four teeth in left maxilla and three teeth in right maxilla.
2. Five root remnants and fractured roots of left molar tooth as revealed by x-rays.
3. Trismus due to injury to external pterygoid and masseter muscles.
4. Anesthesia to right side of face with indications of nerve regeneration taking place.
5. Intraoral scar adhesions to right and left alveolar ridges, obliterating the mucobuccal fold on the left side.

The following treatment was undertaken:

First Operation.—On April 17 removal of root remnants and excision of vicious cicatrix in left lip by intraoral approach completely released the skin of the face. The oral mucous membrane was undermined buccally and palatally. A liberal ridge was obtained by suturing the flap edges and tacking them to the periosteum at the mucobuccal periphery. Healing was uneventful. Three weeks later, with complete healing, the patient was referred for prosthetic restoration (Fig. 12).

On June 2 the patient complained of stiffness in the right cheek with marked trismus. The scar area was sensitive to touch.

On June 4 his face was grossly swollen with exacerbation of symptoms. The patient complained of constant pains radiating to the ear, and he was unable to chew.

On June 5 an abscess of the right cheek was incised and thick pus was obtained. A rubber-dam drain was inserted.

On June 8 the abscess resolved and there was no drainage or swelling.

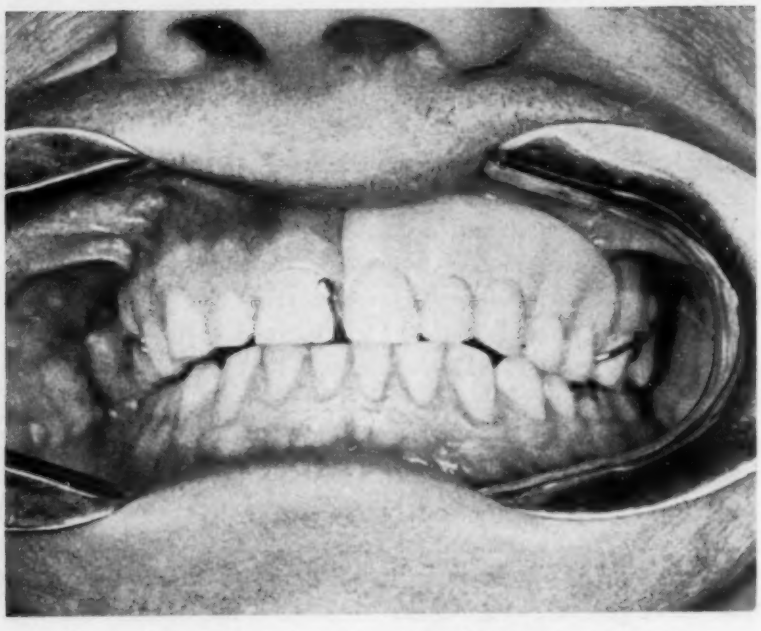


Fig. 12.—Case 2. Prosthetic replacement of missing teeth.



Fig. 13.—Case 2. S-incision in right cheek.

Second Operation.—On July 11, following the Ivy and Curtis technique for relieving scar tension, a Z plastic closure was performed on the mucous wall of the right cheek. The incision was made (Fig. 13) in the form of the letter *S* undermining full thickness flaps. Bands of scar tissue were dissected clear to the undersurface of the skin. The *S* was reversed into *Z* and sutured into place, using catgut sutures subcuticularly and nylon superficially.

Edema followed but was controlled by hot continuous irrigations and systemic penicillin therapy. The nylon sutures were removed a week later. Postoperative treatment after healing consisted of muscle-stretching exercises by means of a spring clothes-pin, and diathermy.

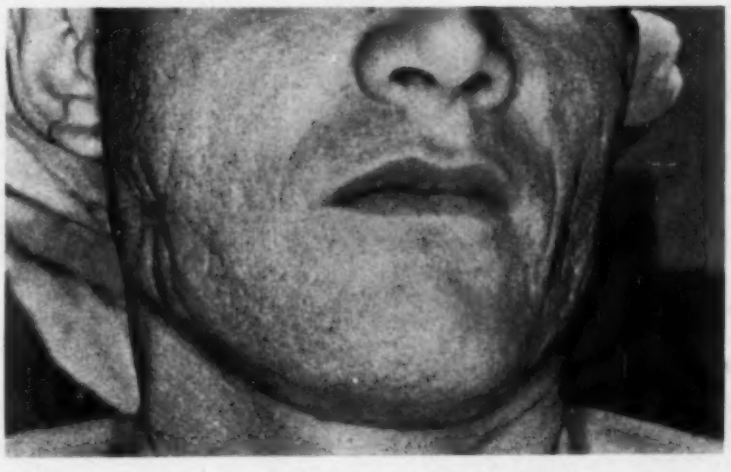


Fig. 14.—Case 2. External radiating scar.

On Sept. 5, 1945, the patient was discharged to duty for the following reasons:

1. All essential operative and prosthetic treatments were considered completed.

2. The patient could open his mouth fairly wide and was able to masticate reasonably well.

3. External radiating scar did not interfere with proper mastication (Fig. 14), and cosmetic correction was postponed.

4. Anesthesia was moderate in severity.

5. Scar on left cheek was fading and was not particularly conspicuous.

CASE 3, FOREIGN BODY, LOCALIZATION

The patient, lieutenant, U.S.M.C., was seen on Feb. 3, 1945. Fracture of left body of mandible was sustained by multiple shrapnel fragments. Examination revealed firm clinical union. A marked undercut was caused by a bulge lingually, due to the initial reduction of the posterior edentulous segment in lingual malposition. A loose sequestrum was found to be in the process of being extruded, with bands of scar tissue and a fistula present.

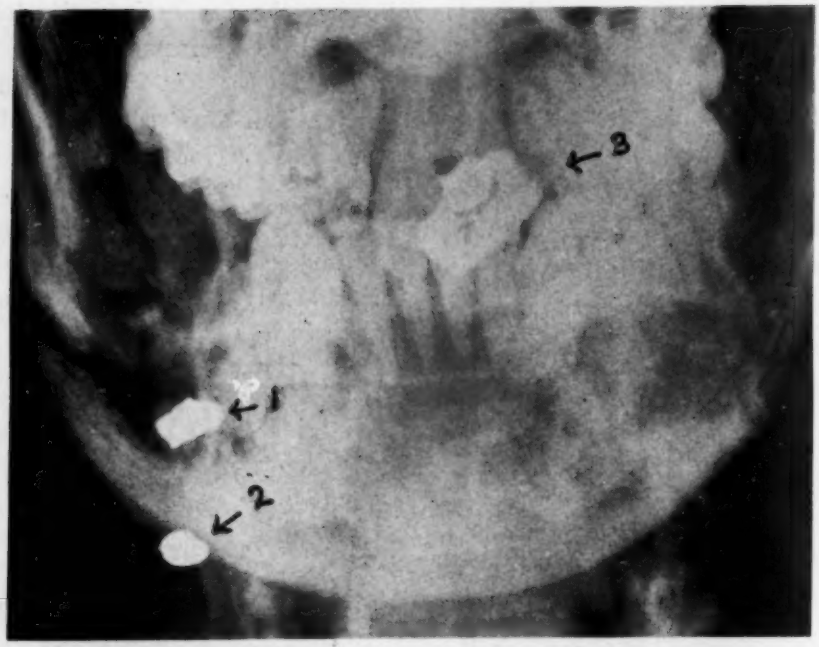


Fig. 15.—Case 3. *1*, Metallic fragment in body of mandible, area of rarefaction, and sequestrum. *2*, Metallic fragment in soft tissue. *3*, Metallic fragment in base of tongue.

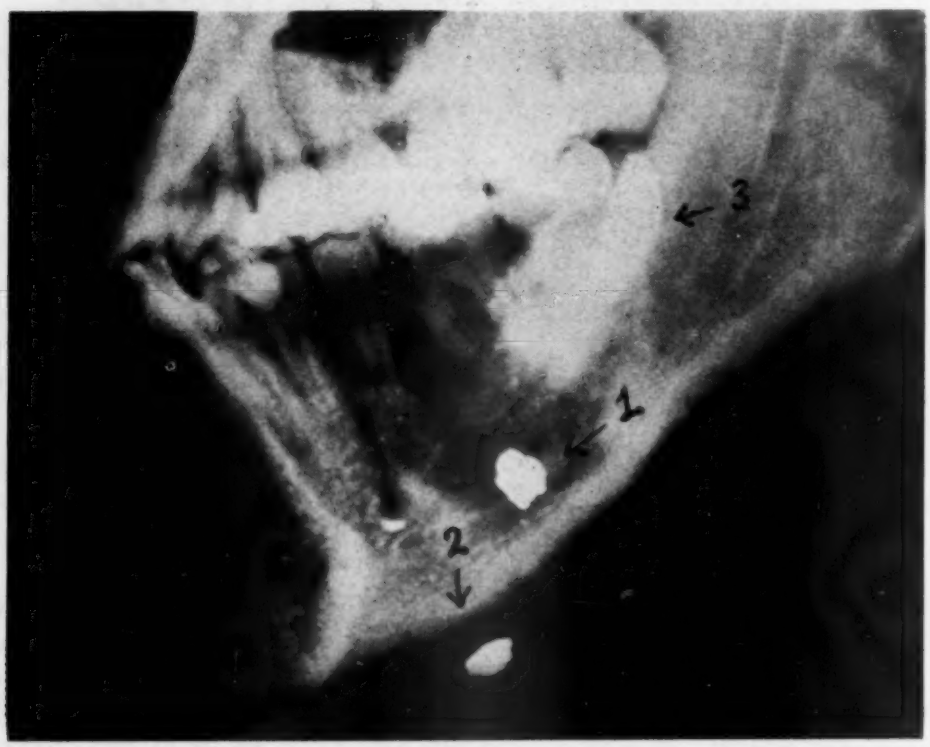


Fig. 16.—Case 3. Lateral jaw view of metallic fragment.

X-ray studies indicated (Figs. 15, 16, and 17):

1. A metallic fragment was imbedded in the body of the mandible surrounded by a rarefied area and sequestrum.

2. A metallic fragment about the size of a pea, seen below the inferior border of the mandible in the region of the premolars was completely imbedded in soft tissues and could not be palpated.

3. A flat fragment about 1.5 cm. long and 0.5 cm. wide was located in the base of the tongue, probably in the hyoglossus muscle.

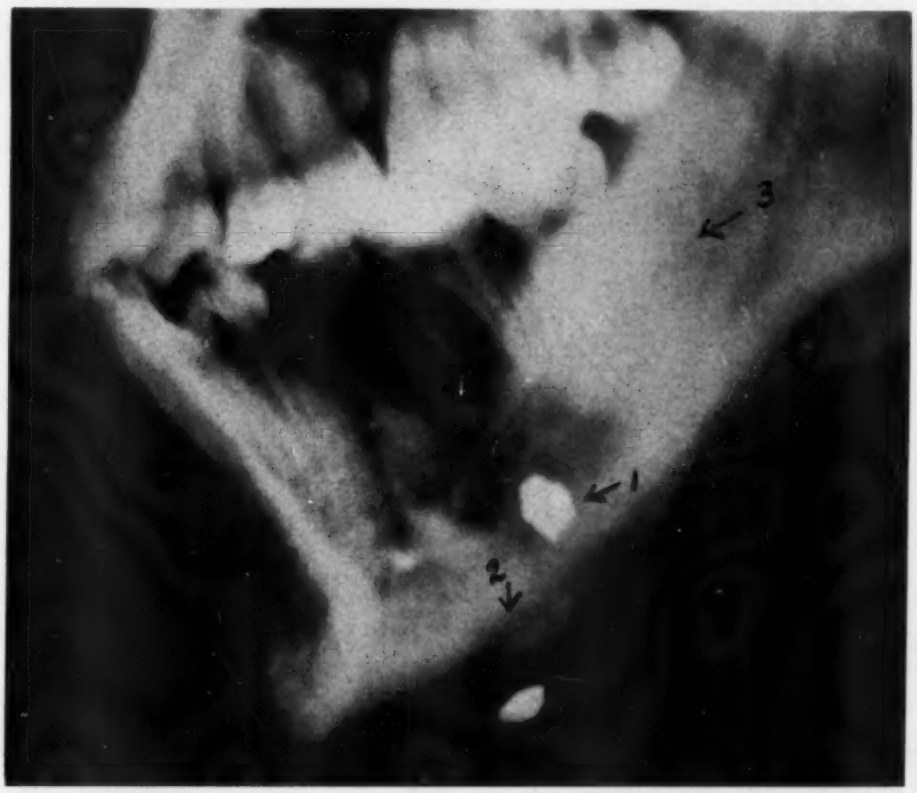


Fig. 17.—Case 3. Note image (3) blurred as patient moved tongue during exposure of film. Compare with Fig. 16. Metallic fragment (2) is below inferior border of mandible imbedded in soft tissue.

Localization.—Technique: Two plates were exposed at approximately the same position and angle. During the exposure of one film, the patient was instructed to move his tongue. In this the metallic image was blurred, indicating that the fragment was located in the base of the tongue.

In most instances, all tissue-imbedded metallic fragments are allowed to remain. Usually the patient is not aware of the presence of such foreign bodies, which have the tendency to become encapsulated. If they tend to become extruded, surgical removal is a simple matter.

Operation.—On Feb. 12, 1945, under regional anesthesia, an incision on the crest of the alveolar bone was made and the underlying bone was exposed. The piece of shrapnel and the sequestrum were removed from the mandible and the area of soft fibrous and granulomatous tissue was curetted. The lingual

bulge was reduced by alveolectomy and the soft tissues were sutured. No attempt was made to remove the foreign bodies in the base of the tongue and below the inferior mandibular border. Six weeks later, the patient was discharged to duty following completion of all essential dental and prosthetic treatments.

CASE 4. PERIODONTOCLASIA

The patient, fireman first class, 25 years old, was under treatment for fungus infection of the skin. After transfer through various medical activities, he was admitted here on March 16, 1945. He was referred to the Dental Department for a coexisting oral condition. His chief complaints were soreness and bleeding of gums for a duration of six years.



Fig. 18.—Case 4. Periodontoclasia: Upper jaw, fibrous hyperplasia. Lower jaw, lymphedematous hyperplasia. Depth of pockets marked. Incision is made connecting markings. Photographed May 25, 1945.

Clinical examination revealed deep periodontal pockets with lymphedematous tissue. In the lower jaw a lush type of hyperplasia was seen, with extravascular cellular elements and fluids easily expressed by pressure (Fig. 18). His teeth, however, were firm.

The upper jaw presented a more chronic stage, resulting in fibrosis.

Full-mouth x-ray examination revealed normal bone structure with moderate horizontal resorption throughout. Alveolar crests appeared apparently normal. Bone recession was noted only in the area of the external alveolar surface of the lower right first incisor.

Diagnosis.—Marginal periodontitis associated with periodontoclasia.

Treatment.—On May 25, complete gingivectomy of the lower jaw was performed. Two weeks later a similar operation was performed on the upper jaw.

The following technique was used:

1. Adequate and effective local anesthesia was used.
2. Depth of pockets was marked.

3. Strip of tissue was removed. Often it is possible to perform gingivectomy by resecting tissues in one or two strips.

4. Underlying granulomatous inflammatory tissue was curetted and scraped down to clean bone, avoiding mutilation of bone. The use of medication for the purposes of asepsis or hemostasis is seldom necessary.

5. Wound was covered with a surgical cement pack.

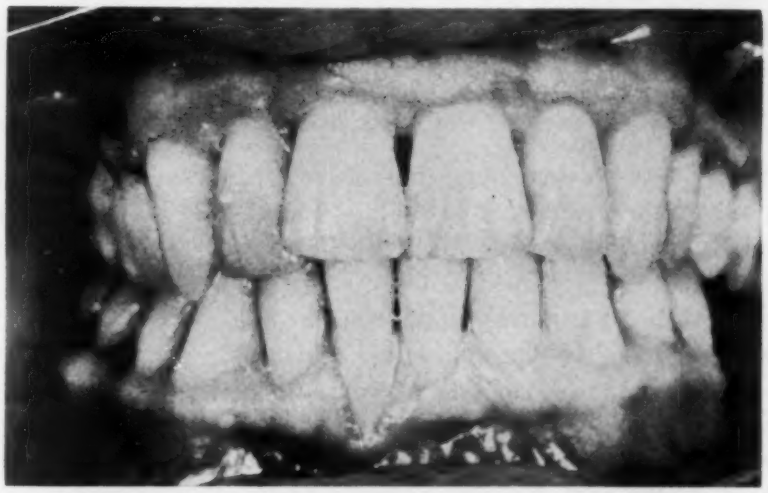


Fig. 19.—Case 4. Postoperative condition on June 25, 1945, one month later than Fig. 18.

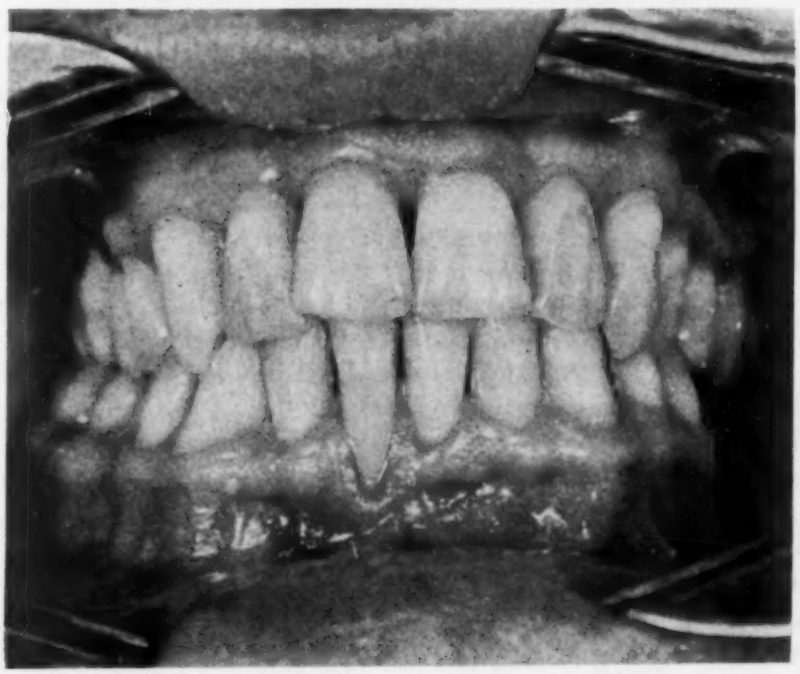


Fig. 20.—Case 4. Postoperative condition on July 6, 1945. Six weeks later than Fig. 18, high spots on incisal edges and cusps were ground for occlusal balance. Occlusal trauma on right mandibular first incisor relieved.

6. Incisal edges and cusps were ground to balance occlusion and relieve the existing occlusal trauma.

7. All essential operative and prosthetic treatment was completed.

The following is the formula for the cement pack used:

Powder.—Zince oxide	120 Gm.	
Finely powdered rosin	120 Gm.	
Liquid.—Eugenol	40 c.c.	
Benzaldehyde	20 c.c.	
Olive oil	0.6 c.c.	(10 drops)

Directions.—A cellophane pad is convenient to use. Mix like cement until the mass is sufficiently thick. Roll in additional powder and knead with fingers to the proper consistency. If desired, the liquid may be tinted pink. If a glass slab is used, xylol will clean it easily.

Success in this treatment depends on two factors: (1) Thoroughness in removing all of the granulomatous tissue is essential. We prefer the use of Crane and Kaplan knives, curettes, and files, but a sharp scalpel and Black's operative chisels may be substituted. (2) The surgical cement pack should be slow setting (15 to 20 minutes) in the mouth. When mixed to a stiff puttylike consistency and rolled in the palms to a ribbon 1 cm. in. diameter, it should not flow or stick to the skin of the operator, yet it must adhere to moist tissues. It is placed in two strips, one for the external surfaces and one for the internal, but pieces may be added when needed. The lips or a moist gauze sponge may be used to mold the pack into place. It is not necessary to use excessive quantities of pack. It should assume the form of a band with rolled margins, avoiding feather-edging. The principle is to cover the "cut surface" and one third of the crowns of the teeth, joining, by gentle pressure, the two crown sections of the pack at the interproximal spaces. This arrangement tends to protect the blood clot in the space between the two incisions, where new tissue is to form. From five days to a week later, the cement is removed with instruments and the wound is gently cleaned with cotton applicators dipped in hydrogen peroxide and flushed with warm saline solution. A fresh pack is then applied and allowed to remain for about another week. No caustics or astringents are used postoperatively.

In three weeks primary healing should be complete, with the formation of new tissue and evidence of healthy granulation and epithelization (Figs. 19 and 20). No other treatment is usually indicated until after three weeks when the patient is instructed to use round toothpicks and other means of stimulation.

Surgical gingivectomy is frequently the method chosen for the elimination of gingival pockets. There are other means available, such as electrosurgery, chemical coagulation, or conservative subgingival curettage. All can be used to advantage and each has its indication.

We have performed a number of gingivectomies on patients whose teeth were firm but where deep pockets and hyperplasia existed. In our experience this is an effective means of eradicating such foci of infection. Elimination of toxic absorption from infected pockets, as a result of gingivectomy, has frequently

resulted in marked improvement of patient's health and a decrease of the sedimentation time. The patients were instructed in the correct toothbrushing technique, interproximal massage, and proper diet to maintain future oral hygiene.

CASE 5. MALOCCLUSION

The patient, private first class U.S.M.C., aged 34 years, complained of inability to chew properly. Palatal tissues in anterior region were constantly irritated and inflammed due to biting pressure. A full complement of teeth was present in the mouth. The maxillary incisors were markedly protruded and

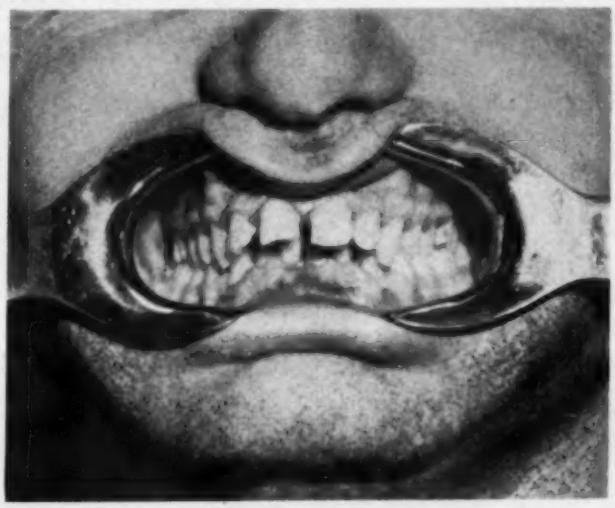


Fig. 21.—Case 5. Preoperative condition. Marked overbite, diasthemas, malocclusion, and closed bite.



Fig. 22.—Case 5. Closed bite and probable pressure into middle ear by abnormal temporomandibular articulation.

mandibular incisors were elongated. Previous treatment had consisted of shortening of mandibular incisors by grinding, but a recurrent elongation had followed. When his mouth closed, the patient had an unsightly overjet (Figs. 21 and 22).

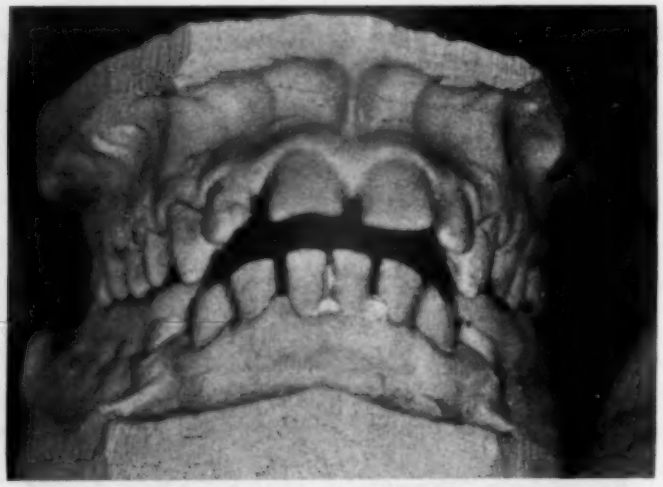


Fig. 23.—Case 5. Casts of mouth showing extent of bite raising.



Fig. 24.—Case 5. Diagnostic bite-raising appliance made of stainless steel heavy wire and methyl methacrylate plastic.

Other complaints were a buzzing sensation in the ears, earache, headache, and dullness in hearing. These symptoms had been attributed to gun-fire concussion and ingestion of quinine in the Bougainville campaign and postmalarial asthenia.

Case study with x-rays and study models indicated the following conditions (Figs. 23 and 24):

1. Closed bite and probable disturbances in the temporomandibular articulation.

2. Gingivitis associated with paradentosis.
3. Poor appearance.
4. Inefficient masticatory mechanism.
5. General physical complications.



Fig. 25.—Case 5. Cast-gold and acrylic permanent splint on model of lower jaw.



Fig. 26.—Case 5. Anterior fixed bridge restoring normal contour and dental arch. Teeth placed in retrusion and bite-raiser is placed in the mouth.

On Jan. 25, 1945, a temporary bite-opening appliance (Figs. 25 and 26), consisting of two acrylic bite blocks fitted over the occlusal surfaces of the patient's mandibular posterior teeth and connected by a stainless steel bar, was constructed. The vertical dimension of the teeth was increased, changing the temporomandibular relations to a near normal position. No elaborate computation was attempted in arriving at the proper extent of bite opening. Our experience indicates that the bite-block thickness is more readily determined by trial and error. We employed the simple test of inducing masticatory fatigue as a clinical guide in the determination of the optimum bite relation in the rest position.

In twenty-four hours the patient was relieved of all aural complaints and chewed comfortably with the appliance in his mouth. His profile was definitely improved. We were convinced that permanent bite opening was indicated.

The maxillary incisors were removed and the protrusion in the alveolar ridge was reduced by alveolectomy.

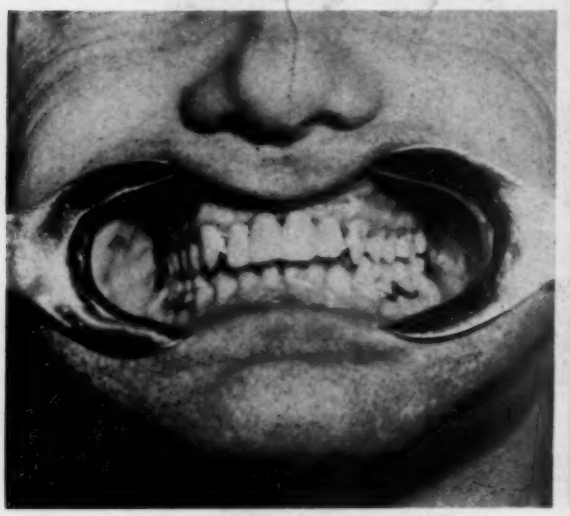


Fig. 27.



Fig. 28.

Fig. 27.—Case 5. Bridge and splint in place. Cusps of lower posterior teeth are built by acrylic removable bite-raising splint fitted over patient's natural teeth.

Fig. 28.—Case 5. Anterior fixed bridge and acrylic bite-raiser worn by patient. Compare facial profile with Fig. 22.

Further treatment by the prosthodontist (Lt. Cyrill Smith [DC], USNR) consisted of the placement of an anterior fixed bridge and a partial mandibular cast gold and acrylic denture, by means of which the bite was raised and the occlusion balanced (Figs. 25 and 26). Essential dental and periodontal treatment followed.

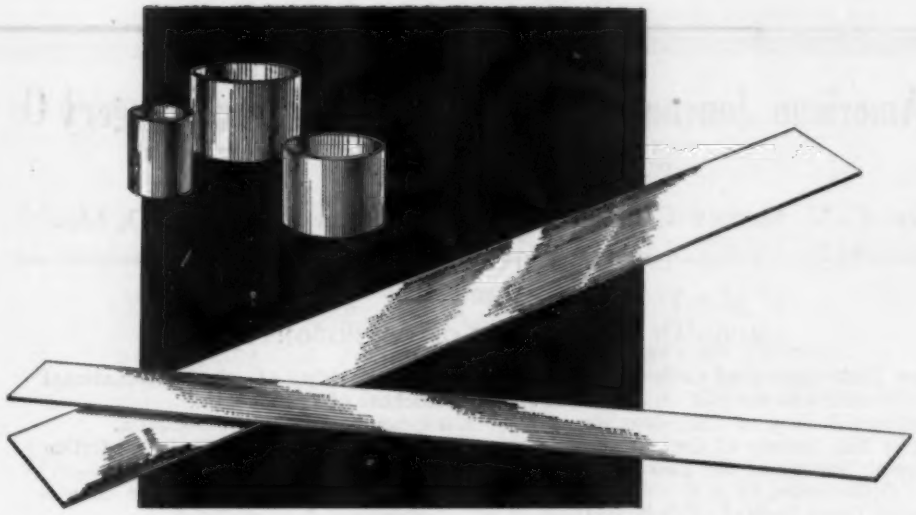
The patient was examined six months later, following continuous use of appliances. He was very well pleased and no longer complained of the previously described symptoms (Figs. 27 and 28). Periodontal disease had subsided as reflected by the improved tone of the gingival tissues.

Grateful acknowledgment is made to Captain Macy G. Martin (DC), USN, Senior Dental Officer, for his advice and assistance in preparing this paper.

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SECOND DENTAL MEDICINE SEMINAR

The Desert Inn, Palm Springs, California ★ October, 1945

Second Annual Dental Medicine Number

From the Division of Dental Medicine, College of Dentistry, The George Williams Hooper Foundation for Medical Research, and the Institute of Experimental Biology, University of California

With Selected Articles from the Second Annual Seminar for the Study and Practice of Dental Medicine

EDITORIAL

WITH this issue the profession is offered for the second time a series of articles selected from the essays presented at the Seminar for the Study and Practice of Dental Medicine. The 1945 session was held in October at Palm Springs, California. The outstanding success of these Seminars and the even greater interest shown in the 1946 session, which is already in an advanced stage of organization, are evidence of the growing interest in Dental Medicine. It is therefore appropriate to summarize some of the potentialities for continued progress in this field.

Dental medicine is not a specialized form of dental practice; it is as broad as the field of science itself. It seeks to bring to the practice of dentistry the application of basic principles of human biology, fundamentals of medicine, and specialized knowledge of the diseases of [illegible] is, in brief, an encourages and lividual rather aches that this ent: establish- nosis, therapy, t the type of expect and to

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Dental medicine is not a specialized form of dental practice; it is as broad as the field of science itself. It seeks to bring to the practice of dentistry the application of basic principles of human biology, fundamentals of medicine, and specialized knowledge of the diseases of oral structures. It is, in brief, an attitude toward practice rather than a specialized regimen. It encourages and requires the consideration of each patient as a complex human individual rather than as a simple problem in oral pathology. Dental medicine teaches that this attitude must permeate every step in the management of the patient: establishment of the etiology and pathology, and determination of the diagnosis, therapy, and prognosis. Only thus can there be brought to each patient the type of attention which the state of scientific knowledge leads him to expect and to which he is entitled.

While the principles employed in dental medicine are fundamental, they are not elementary. In the foreseeable future, as in the past, the greatest interest in the study of these principles will continue to be found among experienced practitioners of dentistry, for it is through repeated observation of the similarities and dissimilarities of disease processes in different individuals that there arises the stimulus to enquiry into basic and underlying causes. It mght, therefore, be predicted that the most favorable opportunities for the study of dental medicine will be found in postgraduate educational programs. In the Seminar,

clinicians and investigators of the many branches and specialties of the health professions meet for open discussion and exchange of information gained through their separate activities.

In the highest professional tradition, the aim of dental medicine is the prevention and eventual elimination of the disorders of oral structures. The fact that this goal is not likely to be attained within our time will be a spur to interest rather than a disheartening influence. As long as there are pathogenic bacteria, as long as there are nutritional deficiencies, as long as there are neoplastic processes, or endocrine dyscrasias, or anemias, or developmental anomalies, or any of the host of human ailments, there will be unsolved problems to which dental medicine may direct its attention.

Advances in dental medicine emphasizing preventive efforts keep pace with the social responsibilities of the modern world. Its principles are likely to be employed most successfully by individual rather than group or community prevention programs of decades to come. The attitude it fosters is susceptible of the best application by individual dentists for individual patients, for its greatest aim is the most constructive consideration of the individual as a whole in relation to his problem of well-being.

Due to the regular and periodic visits of his patient, the dentist occupies a strategic position in the healing professions. With training in dental medicine, he may perform greater services for the health of these patients. His knowledge of the borderline clinical fields will enable him to detect early symptoms and recognize incipient threats to well-being. He will thus better serve his patient and more constructively cooperate with his contemporaries in the many fields of health service. Dental medicine as the spearhead of the drive on prevention of disease may thus contribute to the realization of the destiny of dentistry.

ORAL PROSTHESIS IN THE REPAIR OF WAR INJURIES

Lt. Comdr. Earl Pound, DC, USNR, U. S. Naval Hospital,
Oakland, Calif.

BATTLE casualties involving structures which are normally under the jurisdiction of the dentist present many problems which are far removed from the routine procedures to which the profession has been accustomed. High velocity and explosive missiles tearing their way through the mouth and jaws create injuries to both the hard and soft tissues that are extremely difficult to repair. The scar contractures, and the mucous membrane and other soft tissue losses frequently present a ghastly and almost insurmountable problem.

The early treatment of compound jaw injuries will not be discussed at length in this paper. In brief, conservatism is the keynote of their treatment. The wounds are closed by suturing, fractures are reduced, débridement is limited to grossly devitalized tissues. Questionable teeth and bone fragments are always left in place. All preserved soft tissue, bone, and teeth will be of great value to both the plastic surgeon and the dentist as the case progresses. Another factor of great importance is prompt prophylactic chemotherapy, in adequate dosage. The degree of primary healing and the small amount of sequestration and abscess formation have been a surprise to all since the advent of penicillin and its combined use with the sulfonamides.

The damage to the osseous structures will range from simple fractures to combinations of shattered bone with large losses of substance requiring the placing of intricate bone grafts. The loss of teeth is a factor always felt very keenly by the men. Their subsequent replacement calls for the most exacting techniques and working hand in hand with the plastic surgeon.

The oral prosthetic appliances required in the rehabilitation of these casualties may be generally classified in two groups:

1. *Fixation Appliances.*—Generally used for the reduction of fractures and the immobilization of osseous parts in bone grafting.

2. *Permanent Dental Appliances.*—For the replacement of missing structures and as an aid in plastic repairs to the soft tissues.

FIXATION APPLIANCES

The immobilization of osseous parts is obtained in three different ways: (1) External fixation. (2) Internal fixation. (3) A combination of these two.

Read before the Second Annual Seminar for the Study and Practice of Dental Medicine, The Desert Inn, Palm Springs, Calif., Oct. 8, 1945.

The ideas and views expressed are solely those of the writer and do not necessarily reflect in general the principles of the U. S. Navy.

The early reduction of fractures of both the maxilla and mandible is very necessary. There has been, however, a surprising number of fractures and cases of extensive bone loss for which practically nothing could be done before arrival at our hospital. In most instances this delay has been occasioned as inferred in the adage, "The man is more important than the wound." The saving of life by establishing adequate airways, by the control of hemorrhage, by the treatment of shock, and by the administration of blood plasm, penicillin, and the sulfonamides, has also been of primary importance.

For this reason, many of our cases have had insufficient or no treatment in so far as adequate immobilization is concerned. Those cases in which interdental wiring would suffice have usually been reduced, but in some of these and all the others, we start from the beginning and carry the case through to completion.

It has been the experience of our staff that the first type, *external pin fixation,* is seldom a necessity. Its use is avoided because of the discomfort to the patients, because of the frequency of the failures observed, and because of the ease and efficiency with which internal fixation appliances are worn. Their use is limited to fractures of the edentulous mandible. We also feel that the use of complicated external appliances attached by means of a plaster of Paris cap has little routine value.

For internal fixations two types of splints are used: acrylic, or plastic and metal. Acrylic splints are easily constructed but their use is quite limited. There are many objections to them mechanically although they are also used to advantage in edentulous mouths and to stabilize horizontal fragments of the mandible in conjunction with circumferential wiring. Generally speaking, they are unpleasant to wear and very unsanitary.

Metal splints have been our choice because of their positive action, cleanliness, and extreme flexibility of design. Metal splints may be used for the immobilization of almost any case in which an adequate number of teeth exist in all or even one of the fragments involved.

First.—The immobilization of fractures in which teeth are present in each of the fragments is a relatively simple procedure and in most of these cases interarch wiring may be dispensed with entirely. The cases are usually so well stabilized that the patient is permitted to chew and has every opportunity to keep his mouth clean.

Second.—Those fractures of the mandible in which no teeth are present in the proximal fragment. These cases may be very simply immobilized by placing an adjustable rod into the splint, which is cemented and belted to the teeth of the distal portion; the proximal stump is manipulated into its correct position; the pointed metal rod is inserted through its sleeve and into the soft tissues and bone of the proximal portion. The rod is then locked with a set screw.

Third.—This type is similar to the second except that one good, badly tilted, or unerupted tooth remains in the proximal portion. The only variation is that the rod, instead of being driven on into the bone, is seated securely into the occlusal surface of this remaining tooth. In these cases we have found it

advisable to secure a wire around this tooth and to create a forward tension upon it, thereby eliminating any chance of the tooth drifting and becoming dislodged from the locking pin.

Another type of case that we are unfortunately called upon to immobilize all too frequently exists when much of the anterior portion of the mandible has been shot away and possibly no teeth, or one or two at the best, remain in the lower jaw. In these cases the stabilizing of the mandibular rami is attained by utilizing a splint on the upper teeth and driving the pin downward and backward into the retromolar area of the ramus after it has been properly positioned. The writer has found this technique quite suitable for reducing fractures in conjunction with interdental wiring and also for stabilizing the rami for purposes of bone grafting.

The fixation of parts prior to the placing of bone grafts calls for the greatest amount of ingenuity on the part of the prosthodontist. Most of our cases have been solved by the metal splint, but they are often used in conjunction with interdental wiring, orthodontic appliances, sliding rods, and adjustable jack screws.

Metal splints are not a panacea for all fracture problems but we do believe that their use is indicated in the majority of difficult cases. Once properly applied, immobilization of the parts is assured, stress is distributed evenly on all the remaining teeth, the mouth can be kept clean and the patient comfortable.

PERMANENT DENTAL APPLIANCES

The construction of permanent dental prosthetic appliances to aid in the rehabilitation of battle-scarred mouths presents a challenge which has to be met with an open mind, a will to do, patience, and skill. Appearance, function and clearness of speech are all at stake for these men. The techniques involved, while not entirely new to the dental profession, are somewhat of a specialized nature.

After major plastic revision of the soft tissue and bony structures, the men are ready for permanent dental restorations. At this time there are frequently three major problems to be faced:

1. The preparation of the soft tissues of the mouth to provide an adequate base for our dentures.
2. The replacing of the missing teeth.
3. The restoration of other missing intraoral parts.

These three problems are seldom handled as separate entities. On the contrary it has been found most advisable, when possible, to take care of all three at the same time.

The reshaping of the soft tissues in the mouth has become almost a routine procedure in the treatment of these casualties.

The loss of soft tissue and mucous membrane lining is frequently considerable. The resultant scar contractures create the loss of the normal buccal and labial sulci. It is commonplace to find the muscles of the face drawn inwardly and connected directly with the roof or the floor of the mouth, with no intervening sulcus in which to terminate or fit a denture. The patient's facial expres-

sion and speech are greatly affected. This condition may be corrected by surgically releasing these scar bands, thereby creating an artificial sulcus. These newly cut surfaces are then lined with a split skin graft to prevent the formation of more scar tissue. All this is accomplished by close cooperation with a plastic surgeon.

The replacing of the missing teeth is by far the most difficult phase of our work because of the freakish mechanical problems created by high velocity missiles. Frequently all of the anterior teeth are destroyed with much of their supporting structures. Few posterior teeth remain. The leverage problem is severe and this invariably calls for stress-breaking appliances.

Another common problem is the loss of all the teeth on one side of the jaw with a continuous row intact on the other. These unilateral cases present considerable bone loss and invariably have close bites on the good side, which increase the problem of stabilization.

A frequent complication in our restorative work is that teeth have to be replaced before plastic revision can be completed about the external mouth. This is done so that the plastic surgeon may judge more accurately true facial contours as they will finally exist. Because of a limitation of mouth opening, full-sized trays cannot be used. This calls for sectional impressions and assembled models which add to the time consumed and the error hazard.

Unfortunately we have some cases in which the mouth is so badly contracted and distorted that it is necessary for the plastic surgeon to enlarge the orifice before any adequate intraoral work can be started. One such case has lost the anterior third of the tongue, two-thirds of the body of the mandible. Most of the palate has been destroyed, leaving oral openings into the sinuses and the nares. Only two upper and two lower teeth remain. This case will call for several intraoral skin grafts, a mandibular bone graft, and the replacement of the missing teeth, together with an obturator to take care of the palatal defects. While this operation is not a part of prosthetic replacements, it is a necessary step to their completion. It will give you a better insight into the severity of our problems and help you to realize that the war will not be over for some of us for a long time.

The replacing of intraoral parts, other than teeth, includes the restoration of portions of the palate and the closing off of sinus defects and cystic cavities. This is usually done with obturators of one type or another, and they may be attached directly or indirectly to the dentures. Each case has to be worked out as the occasion demands. There are no set rules to follow. Plastic is the material of choice. Both the hard and the flexible plastics have their place, although the flexible is avoided because it absorbs odors and is, therefore, unpleasant.

In conclusion, there are a few thoughts that would be well to extend to you. First it must be borne in mind that while fractures are reduced, bone grafts are inserted to replace lost structure and the soft tissues of the mouth are reshaped to more natural contours, the finished result still bears the unmistakable effects

of trauma and loss. Many thousands of these patients will soon be thrown upon the dental profession and their problems will be severe and their demands exacting. A second factor to which one must be alert is that most of these men will be carrying bits of metal, coral, gunpowder, and such about the head and neck for the rest of their lives, and these foreign particles are bound to give rise to symptoms that could easily be improperly diagnosed. Periodic allergies, illnesses, and states of depression will affect some of these men for many years. When these men come to you for attention, they do not seek sympathy, they seek the finest professional service. Study their problems; study their histories; be thorough. They are deserving of the very best we have to offer.

CLASSIFICATION AND TREATMENT OF THE RHEUMATIC DISEASES WITH SPECIAL EMPHASIS ON INFECTIOUS AND RHEUMATOID ARTHRITIS

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OUR knowledge concerning the diagnosis, pathology, and treatment of the rheumatic diseases has gradually increased during the past fifteen years. During this period of time both the laity and the medical profession became more and more aware of the tremendous importance of this group of diseases as a social and economic problem. It has been estimated that over 300,000 persons are rendered unemployable annually in the United States and that the cost of the disability may exceed two milllion dollars. A sound basis for the reorganization of the arthritis program was the recognition of the need for hospitals and clinics devoted to the study of the special subject of arthritis. Throughout the United States, committees associated with medical schools and institutions devoted to research have been organized for the purpose of group stimulus in the study and treatment of patients with joint diseases. The results of the endeavors of these groups have contributed much to a better understanding of the rheumatic diseases.

In 1934, a society was organized for the study of rheumatism and is now officially recognized as the American Rheumatism Association. One of the subjects that was frequently discussed by its members soon after organization was the need for a better definition and differentiation of the various diseases of the joints. After several years of study and after having undergone revision several times, the American Rheumatism Association in 1941 finally adopted a classification of the rheumatoid diseases. This may be recorded as follows:

1. Specific infectious arthritis (organism known).
    a. Gonorrheal arthritis.
    b. Pneumococcal arthritis.
    c. Tuberculous arthritis.
    d. Brucellosis arthritis.
2. Arthritis of rheumatic fever.
3. Rheumatoid arthritis (synonyms: atrophic, proliferative and chronic nonspecific infectious arthritis; Still's disease; Marie-Strümpell spondylitis).
4. Osteoarthritis (synonyms: degenerative joint disease; hypertrophic senescent arthritis).
5. Arthritis of immediate traumatic origin.
6. Arthritis of gout.
7. Arthritis of neuropathic origin (Charcot's joint).
8. Neoplasms of joints.
9. Miscellaneous forms (arthritis associated with other diseases).

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BASIC PROCEDURES IN DIAGNOSTIC SURVEY

Before therapy can be outlined for any given case of diseases of the joints, it is important for the physician to diagnose and properly classify his patient's illness. The basic examinations executed in our clinic are the following:

1. A compete history is taken, with special attention to familial predisposition to the rheumatoid diseases, dietary abnormalities with emphasis on a possible deficiency in vitamin C, exposure to extreme and sudden changes in temperature and moisture, repeated attacks of "low-grade infections," trauma to joint tissues and the mode of onset of the illness, whether or not there is pain, swelling, redness, disability, and progression of symptoms from one joint to another, and the duration of these symptoms.

2. A thorough physical examination is made, including observation of the characteristic changes of the joints and their degree of disability; tophi on the ears; nodules in the skin, subcutaneous tissues, or periosteum; a search for foci of infection; and abnormalities of the heart, lungs, or kidneys.

3. The laboratory procedures should include urinalysis, complete blood count, rate of sedimentation of the red blood cells, Wassermann test, and estimation of plasma ascorbic acid and blood uric acid.

If possible, it is desirable to have: (a) A roentgenologic study of one or more of the involved joints. (b) A photograph of one or more joints. (c) Removal of synovial fluid for diagnostic study.

CLINICAL CONSIDERATIONS

With the afore-mentioned facts in mind, this lecture will be limited to a discussion of the diagnosis and treatment of infectious and rheumatoid arthritis.

Specific Infectious Arthritis.—Gonorrheal rheumatism occurs during the course of active infection of the urethra with the gonococcus. The joint manifestations usually make their appearance after the urethral infection has become well established. Usually there is sudden onset of swelling in a single joint. This is accompanied by excruciating pain, redness of the skin, and disability of the affected member. The diagnosis may be confirmed by obtaining cultures of the gonococcus from the synovial fluid.

It is important in the treatment of this disease first to control the pain. This may best be accomplished by the administration of one of the opium derivatives. Some relief may be afforded by applying mechanical supports for the purpose of preventing movement within the joint. The gonococcus shows a high degree of sensitivity to both the sulfonamides and penicillin. Thus, specific therapy which results in rapid disappearance of the infection may be offered the patient.

Tuberculous arthritis is another example of disease of the joints due to a specific organism. This frequently is monarticular in distribution. The hip joint, the vertebrae, or possibly a group of small bones such as the wrist may be the site of infection. There is localized heat, swelling, disability, and excruciating pain. In this illness the need for a careful general physical exam-

ination is emphasized because of the necessity to seek out a primary focus of tuberculous infection elsewhere in the body. The treatment is mainly one of rest and general supportive measures.

Another type of joint swelling appears on occasions in a patient with tuberculosis in which the tubercle bacillus itself cannot be isolated from joint structures. This arthrosis is called tuberculous rheumatism and is thought to be an allergic manifestation and one in which the marked bony destruction seen in tuberculous arthritis does not occur.

During the course of illness of other infectious diseases, arthritis may appear as a complicating factor. In lobar pneumonia, scarlatina, meningitis, subacute bacterial endocarditis, Haverhill fever, brucellosis (undulant fever), and many others there may appear swelling of one or more joints. Such swelling, however, is arthrosis, rather than true arthritis.

Arthritis of Rheumatic Fever.—It is becoming more apparent that rheumatic fever is a contagious infectious disease. There have been rather widespread outbreaks in various Army camps. It is possible that a type of hemolytic streptococcus is the etiological factor. The onset of illness usually is ushered in with sore throat, followed in three days to two or three weeks by swelling of the joints. A single joint may show swelling, pain, redness, and disability. Characteristically, these symptoms may abruptly subside only to appear in some other joint. This migratory character of joint involvement is a feature of rheumatic fever.

Evidence of active illness may be apparent over a period of months. The swelling of the joints may subside and the pain lessen, but the persistence of fever, rapid sedimentation rate, and the occurrence of audible changes in the character of the heart sounds are evidences that an inflammatory process is in progress in the mitral valve.

There is no specific treatment for rheumatic fever. Absolute bed rest, large doses of sodium salicylate and ascorbic acid, and a well-balanced diet are indicated.

Rheumatoid Arthritis.—In rheumatoid or atrophic arthritis, changes take place not only in the bony and hard substance of the joints but the soft tissues as well. The swelling, the increase in local heat, the redness of the skin observed by the physician, and the pain and stiffness suffered by the patient are caused by an active inflammation of the synovial membrane, the capsule, and the surrounding soft tissues of the joint.

In a few cases of rheumatoid arthritis, a single joint may be involved early in the course of illness. In most instances, however, the characteristic distribution is symmetrical and one in which many joints are affected. The sites of election for the onset of the disease may be frequently found in the interphalangeal and the metacarpophalangeal joints of the hands. The disease may progress to include the joints of the feet, ankles, knees, hips, elbows, shoulders, and possibly the spine.

Widespread distribution may take place rapidly over the period of a few days during which time the patient is acutely ill, has fever, loss of appetite, and rapid loss of weight, and suffers intense pain. In contrast to this is the

illness ushered in by soreness of a joint, transient pain, and swelling that progresses slowly over a period of weeks or months.

It is of considerable importance that these two extremes in the onset of illness be recognized so that they may be properly treated. Only too often, patients with fulminating rheumatoid arthritis have been placed in bed and given analgesics to relieve pain without thought on the part of the physician as to the degree of disability that may ensue. Under these circumstances, as the patient lies in bed, the joints gradually stiffen and eventually become ankylosed, due to the inherent proliferative character of the pathologic process. He is unable to use his hands, he cannot bend the elbows or knees, and proper function of the feet is lost because the weight of the blankets has caused them to be rotated outward and fixed in this position.

The treatment of the patient with rheumatoid arthritis should be carefully planned. Frequently these patients show evidence of improper nutrition and are underweight. A well-balanced diet sufficiently high in calories should be prescribed for the purpose of adding weight. Fresh vegetables and the juice of citrus fruits should be included for their content of vitamin C and minerals. It is one thing, however, to prescribe a diet and another to have the patient eat the food brought to him. Individuals with a chronic illness do not desire food and seem content to "pick" at it. The physician, therefore, must persist in encouraging the patient to eat.

Anemia is frequently present in patients with rheumatoid arthritis. Unfortunately the anemia is refractory to such antianemic principles as large doses of iron or liver extract. It is advisable, then, to transfuse the patient with sufficient amounts of whole bloood to bring the erythrocytes and their hemoglobin content to within normal values.

It has been shown by Rinehart and others[1] that the plasma ascorbic acid content is reduced in patients with this disease. In order to remedy this deficiency it is necessary to supplement the diet with ascorbic acid in the form of tablets in dosages of 50 to 100 mg., two or three times daily.

The orthopedic surgeon and a competent physical therapist give invaluable aid in the management of the swollen, painful, deformed joints. This type of treatment is of considerable importance and could be discussed at great length. Suffice it to say here that casts, properly applied to permit rest of a joint and reduction of muscle spasm, afford relief of pain to the patient. However, these casts should be so constructed as to permit passive motion in the joint twice daily to prevent the formation of adhesions and its consequent bony ankylosis.

Since 1925, there has been slow but gradually increasing interest shown in the use of colloidal gold in the treatment of rheumatoid arthritis. Injection of 50 mg. of the solution* at intervals of one week is the recommended dosage. Treatment must be persisted in until the patient receives from 60 to 100 injections. A satisfactory response will occur in about 80 per cent of the cases so treated and will be recognized by alleviation of pain and swelling, and by a return to normal of the sedimentation rate. It must be emphasized, however,

*Gold sodium thiosulfate, G. D. Searle & Co., or gold sodium thiomalate (myochrysine), Merck & Co., Inc.

that this drug is toxic and that mild to severe untoward reactions will be observed in about 50 per cent of cases so treated. Itching of the skin, erythematous lesions, or severe exfoliative dermatitis may appear. Nephritis, purpura hemorrhagica, and granulocytopenia have been recorded.

Very little has been said in this report concerning the role that focal infections may play in the cause of rheumatoid arthritis. Too often patients are seen who have had one or more teeth extracted with the hope of altering the course of the arthritis, only to find that the inflammatory process in the joint is unyielding. Whereas formerly much emphasis was placed on the "focus of infection" theory of arthritis, most rheumatologists now are of the opinion that there must be some other etiologic or precipitating factor. However, if there is evidence of an apical abscess, this infection should be properly treated as a hygienic precaution, in an endeavor to improve the general health of the patient.

SUMMARY

Some of the contributions to the subject of arthritis over the past fifteen years are reviewed. A revised classification and differentiation of joint diseases, as adopted by the American Rheumatism Association, is presented. The general medical management with regard to diet, physical therapy, and the use of newer drugs such as sulfonamides, penicillin, and colloidal gold is stressed in contrast to the former popularity of the "focus of infection" theory which is relegated to a less important role.

REFERENCE

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THE DENTIST LOOKS AT THE PATIENT

By a Neurologist

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SIMPLY by looking at the patient who sits in the dental chair, the dentist may recognize some neurologic affections which leave their imprint on the patient's face. No questions need be asked; no undressing, no physical examination is necessary—just discerning observation of the face is required. Early recognition of such neurologic affection may prove highly valuable from a general medical standpoint and may influence the whole plan of the dental work. Generally speaking, the patient sees his dentist more often than he sees his physician. By drawing the patient's attention to his neurologic condition, which usually develops very insidiously, the dentist can render the patient a great service, all the more so since much depends on early recognition of the symptoms.

Lantern slides demonstrating some neurologic conditions easily recognizable on the face of the patient were shown, and the symptoms depicted were briefly discussed:

1. *Encephalotrigeminal Hemangioma.*—Congenital enlargement of the skin vessels of the face may be limited exactly to the cutaneous area of the first or second division of the trigeminal nerve. This distribution shows the neurogenic influence in these congenital abnormalities of vascular development. If such a patient complains of paresthesias or twitching in the contralateral extremities, or of convulsions, it indicates that the vessels inside the skull are affected in the same way as the vessels of the skin. This is a serious condition which may require surgical intervention.

2. *Progressive lipodystrophy* is a very striking and rare condition due to disturbance in the central regulation of fat distribution in the body. The face and the upper part of the body become devoid of fat; the patient looks emaciated from the face to the waist, but enormous masses of fat have accumulated below the waistline.

3. *Facial Hemiathrophy.*—Fat and subcutaneous tissue shrink slowly in one-half of the face, particularly near the midline. The hair and skin may be involved too. This condition is also due to a disturbance in central trophic regulation.

4. *Paralysis agitans* is a chronic, relentlessly progressive disease, due to slow degeneration of some central parts of the brain. There is a characteristic stiffness, weakness, and tremor of the body. The head is bent, the face is ex-

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pressionless, masklike. The facial muscles are immobile, the palpebral aperture is widened, the blinking of the eyelids is infrequent. The patient stares ahead, and is slow in articulation, mastication, and deglutition. The speech is slurred, monotonous. A smiling expression of the face develops slowly, and is unusually protracted.

5. *Encephalitic Parkinsonism.*—Here the face of the patient resembles that seen in paralysis agitans. Excessive discharge of saliva is more common. The skin of the face has a greasy appearance. This parkinsonism may develop many years after the acute stage of epidemic encephalitis. Such an epidemic swept the whole world in 1916 to 1919. There may be a complete alopecia as a sequela of this encephalitis.

6. *Pathologic Laughing and Crying.*—In diffuse lesions of the brain due to various causes, the patient may suffer an impairment of his voluntary control over his emotional reactions; thus, exaggeration, prolongation, and deepening of the normal emotional response take place. The patient laughs or cries on the slightest provocation or without any recognizable cause. He is unable to stop once having begun to laugh or to cry. Sometimes the reaction is reversed: he laughs at bad news, and vice versa.

7. *Herpes Zoster.*—This is a vesicular eruption mostly due to an acute infection of the first sensory neuron and of the corresponding skin. The first division of the trigeminal nerve is often affected, due to involvement of the corresponding part of the gasserian ganglion. The postherpetic neuralgia which often is a sequela of this ophthalmic zoster may be very distressing—especially in elderly people—since it shows high resistance to treatment.

8. *Angioneurotic Edema.*—In this condition a transitory edema occurs and recurs in the face, due to increased permeability of the capillaries; it may become very intensive and extensive. The respiratory pathways may be obstructed and create a life-endangering situation. The disorder is probably allergic in character. Injections of adrenalin are helpful.

9. *Oculogyric Crises.*—Here sudden attacks occur in which the patient has a spasm of the muscles of the eyeballs. Both eyes deviate, usually upward, and sometimes the head is retracted. The attack may last a few seconds or for hours. These recurrent attacks are sequelae of epidemic encephalitis and may occur in patients who otherwise have only mild sequelae of this disease.

10. *Horner's Syndrome.*—This consists of (a) slight ptosis—the upper lid droops; (b) myosis—the pupil of the affected eye is smaller than that of the healthy eye; (c) enophthalmus—the eyeball is retracted. This syndrome is due to an affection of the sympathetic innervation of the eye. The lesion lies usually in the cervical sympathetic chain.

11. *Oculomotor Nerve Palsy.*—The patient shows marked ptosis, the eye deviates outward; it cannot be moved upward, downward, or inward. The pupil is dilated. The oculomotor nerve which controls these movements is often affected either by local lesions acting directly on the base of the brain (syphilis, vascular lesions, meningitis) or by remote lesions like brain tumor of any location.

12. *Tabes Dorsalis.*—Here paralysis of the oculomotor muscles, sometimes transient, occurs in the early stage of the disease. If both oculomotor nerves are affected, a typical "tabetic face" results: mild bilateral ptosis, compensatory overaction of the frontal muscles, both eyes are turned outward. The pupils in this disease are unequal in size, have irregular shape, and do not contract when they are exposed to light. Tabes dorsalis is of interest to the dentist because in this disease spontaneous shedding of teeth, analgesia, and pain in the trigeminal area can occur. Such pain may simulate a purely dental affection.

13. *Abducent Palsy.*—The eye deviates inward and cannot be moved outward. The causes of this palsy are, generally speaking, the same as those of oculomotor palsy.

14. *Facial Palsy.*—All the muscles innervated by the facial nerve are paralyzed if the lesion is located in the nucleus or peripherally. The paralysis spares the frontal muscle if the lesion is above the nucleus—in the pyramidal tract between the nucleus and the cortex of the brain. In such a purely pyramidal lesion, the facial muscles which do not respond to voluntary stimulation may still respond to emotional stimulation—for instance, if the patient laughs. If the lesion is in the thalamus, the reverse may happen: the affected side moves normally on conscious effort, but lags behind when the patient laughs. If a peripheral facial palsy has recovered only incompletely, contractures of the formerly affected muscles and so-called "mass movements" may occur: the patient—for instance—when asked to close his eye, automatically pulls up the corner of his mouth at the same time.

15. *Facial Tic and Torticollis.*—Both affections consist in repetitive, monotonous, involuntary movements, of all the facial muscles or of part of them, and of the neck muscles, respectively. Both afflictions (a) may be purely psychogenic and thus an expression of an obsessive compulsive neurosis; (b) may originate as a normal reaction to a local irritation, persist when the irritation is removed, and thus degenerate into a habit; (c) may be due to an organic disease of the central cerebral ganglia, such as encephalitis or primary degeneration.

16. *Myasthenia.*—The patients—mostly women—show a weakness of the muscles innervated by different cranial nerves. Ptosis often appears first. The onset is insidious, the course fluctuating. Pathognomonic is the abnormal fatigability of the affected muscles. This can be easily demonstrated by asking the patient to close his eyes repeatedly, or to count. Patients develop difficulties in chewing, swallowing; speech becomes slurred, nasal, hoarse. This is a very serious affection. The site of the disease is at the neuromuscular junction. Prostigmine, acting as a metabolic substitute, brings dramatic but transient relief. Greatest caution should be exercised in all dental work on such patients. Sudden death may occur, especially on exertion. A sufficient amount of prostigmine should be given before any emergency dental operation is undertaken, and a physician should be consulted without fail.

17. *Trigeminal Palsy.*—In a unilateral palsy of this nerve, there is atrophy and weakness of the muscles of mastication on the affected side. When the patient opens his mouth, the jaw deviates to the affected side, due to the unopposed action of the external pterygoid muscle on the healthy side.

18. *Trigeminal Neuralgia.*—This disease was formerly and characteristically called "epileptiform neuralgia." Paroxysmal and brief attacks of severe pain occur in the trigeminal area, mostly in the second and third division. Touching, washing the face, talking, or masticating may provoke such an attack. During an attack there is often a reflexive cramp of the facial muscles. Even after long duration of the neuralgia, objective changes with regard to motility, reflexes, or sensibility in the trigeminal area cannot be demonstrated. The exact etiology of this affection, even its point of origin, is not definitely known.

19. *Symptomatic Trigeminal Neuralgia.*—Here pain in the trigeminal area is produced by a gross organic lesion of the brain. The pain is usually persistent and is accompanied by—though sometimes mild—objective signs of cerebral involvement. The underlying lesion may be tabes, multiple sclerosis, local meningitis, bony exostosis, anomaly of vessels, brain tumors—especially tumors of the cerebellopontine angle. The latter are of special importance since they are not very uncommon and are amenable to surgery. These tumors have a very insidious onset and a slow course. Deafness and tinnitus, followed later by facial and trigeminal nerve palsy, are characteristic. Pain, such as observed in trigeminal neuralgia, may occur. The slow-growing tumor produces these signs by gradual displacement of the eighth, seventh, and fifth cranial nerves at the angle between pons and cerebellum.

20. *Hypoglossal Palsy.*—Unilateral palsy of this nerve, always a symptom of a grave organic nervous disease, produces wasting of the muscles of the corresponding half of the tongue. On protrusion, the tongue deviates to the affected side, due to the unopposed action of the genioglossus muscle of the healthy side.

21. *Progressive Bulbar Paralysis.*—The tongue shows marked atrophy and fibrillations. The muscles innervated by cranial nerves other than the hypoglossal are also involved, leading to difficulty in speaking, masticating, and swallowing. This affection is due to a primary degeneration of the nuclei of the cranial nerves. It is one of the most distressing diseases of the nervous system for which there is no known therapy.

THE PHYSIOLOGY OF WATER IN HEALTH

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IT IS desired to review here some of the facts about water, and to call attention to the great contribution water makes to the life of a healthy individual. By so doing, it is hoped to encourage more interest in the most abundant and most useful commodity at our disposal. From birth until death, water is so omnipresent in the daily life of the baby, the child, the adolescent, and the adult, that he is inclined to take it for granted, and seldom stops to think what our world would be like without water.

A topographical survey reveals that three-fourths of the earth's surface is covered by water. Geodetic engineers have reckoned that if the earth's surface were leveled, the oceans at the present time contain enough water to cover it to a depth of nine thousand feet. This does not include the rivers, lakes, nor inland seas, nor the subsoil water that feeds the springs and artesian wells, nor the water that floats above in the clouds. One cannot avoid the conclusion that any substance which so greatly exceeds all others in the world must make a tremendous contribution to life as it exists on earth.

The truth is that there can be no life, plant or animal, without water. Protoplasm, living substance, which Huxley has called the "physical basis of life," is 75 per cent water. It is a mixture of crystalloids and colloids in water. The younger and more active the protoplasm, the greater the amount of water it contains. Water cannot furnish any energy as such, but it does enter into all the changes which determine the transformation of matter and energy in the organism. Water is mobilized and shifted about in the body more frequently than any other substance. No other chemical compound serves so many vital functions as water.

A man can go without food for a period of fifty days without serious damage if he has water. If deprived of water, desiccation develops, wrinkled skin, dry mucous membranes, cracked tongue, high fever, severe acidosis, delirium, and a horrible death within a few days.

Research in the chemicophysical field, in biochemistry, and in chemistry, seldom stresses the major part that water has served in obtaining the final results. We are in the habit of thinking of water as H_2O. Recent investigation has proved that this is not the true chemical structure of water. Ordinary rain water is a mixture of monohydrol, H_2O, 21 per cent, dihydrol, H_4O_2, 50 per cent, and trihydrol, H_6O_3, 21 per cent. In addition, considerable amounts of $(H_2O)_4$ and $(H_2O)_6$ are also present. There must be added to this, traces of heavy water, deuterium oxide, 1 to 8,000.

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A consideration of some of the contributions water makes to the health of the individual reveals:

First, that the great amount of water in the tissue cells structurally renders the tissues plastic and incompressible. The cell is like a rubber bag filled with water; it is plastic and resilient to applied pressure. Muscle tissue is 75 per cent water, whole blood is 80 to 85 per cent, plasma 91 per cent, brain tissue 80 per cent, bone 25 per cent, and fat 20 per cent.

Second, water serves as a lubricant between the surfaces of the viscera. Pericardial fluid facilitates the movement of the heart in the pericardial sac. The same is true of the fluid in the pleural cavity during the excursions of the lungs in inspiration and expiration. Again, in the abdomen the peritoneal surfaces, moistened by water, permit the intestines to move about freely. Likewise, the movements of the joints are aided by synovial fluid. The central nervous system is surrounded by cerebrospinal fluid.

Third, more substances will dissolve in water than in any other solvent. It is the universal medium for the infinite variety of metabolic activity continually going on within the cells.

Fourth, water is a powerful ionizing solvent. By virtue of this property of water, an osmotic force is produced in the tissues which controls the distribution of fluids and solutes within and without the cell membrane. Cations and anions are released. This ionization is the first step in almost all of the reactions which take place within the tissues. Metals, acid radicals, and bases are thus made available to the cells, and as a result the cells are supplied with the materials to carry on their own specific metabolic activity.

Fifth, water is the vehicle which transports food material, internal secretions, and waste products from their sources to the point where they are disposed of by the tissue cells. The scope, the importance of this function of water may not be apparent until one considers secretion, absorption, and the circulatory system. In the latter case, Nature has provided man with the most perfect transportation systems in the universe. We must not forget that most of the water in the body is not static in the tissues, but that it keeps moving all the time. For example, the systolic thrust of the left ventricle forces the blood under pressure into the aorta, and the elastic recoil of the vessel walls, numerous valves, and the contraction of muscles keeps it moving onward until it has completed the circuit. Rowntree, by the dye method, computed the quantity of blood in the body to be equal to one-twelfth of the body weight. With the heart rate of 75 per minute, and the systolic output of the heart at a minimum of 80 c.c., 6 liters of blood, the total quantity in the body, would pass through the heart every minute. This is under basal conditions. Actually the volume is much greater. As this blood leaves the heart it enters a vascular system which spreads out in the shape of two cones with their bases together. The apex of one cone is at the left ventricle, the other is at the right auricle. The aorta supplies innumerable diverging smaller vessels which, as they approach the base of the proximal cone, end in the capillary bed, the cross section of which is eight hundred times greater than that of the aorta. The blood continues into

the second cone, converging to its apex, which terminates in the vena cava at the right auricle. It is to and from this capillary bed (the broad bases of our cones), that water conveys the essentials for the cells' metabolism.

Krogh, in his article on "The Anatomy and Physiology of the Capillaries," has estimated that, if put end to end, the capillaries in a man's body would be sixty-two thousand miles long. He also estimated that the surface area of this capillary bed was one and one-half acres. To visualize the total volume of blood spread out over such a vast area, separated from the tissue cells by a single layer of endothelium, at least once every minute, is something to marvel at. It is true that blood is not evenly spread out over this great surface, but under vasomotor control is shifted by contraction of arterioles supplying one part of the capillary bed, and dilatation of some other part to meet the increased demand for supplies.

Water is the vehicle for the active agents in the digestive juices. Saliva, gastric juice, bile, pancreatic juice, and intestinal juice all together add up to from 7 to 8 liters; 2 gallons of fluid secreted into the alimentary canal every twenty-four hours. Added to this is the ingested water in the amount of 3 liters per day. This large volume of water, carrying the digested products, moves back into the blood stream through the intestinal mucosa.

The skin contains some two million coiled tubular sweat glands which discharge from ½ to 1 liter of fluid onto the surface of the body every day. This is greatly increased in the regulation of body temperature, when either internal or external temperature is increased.

A study of the structure and functions of the kidney still further emphasizes the manner in which water serves as a conveyor, in this case, of waste products to be excreted. Within the kidneys there are three million nephrons, each consisting of a malpighian body and a uriniferous tubule. The malpighian body is formed by a coil of blood vessels, the glomerulus, and a cuplike sac, Bowman's capsule. This latter is continuous with the uriniferous tubule. These tubules are very tortuous in their course from Bowman's capsule in the cortex of the kidney until they reach the collecting tubules which empty into the pelvis of the kidney. It has been estimated that these tubules all together would be forty-five miles in length.

Every twenty-four hours of a man's life there is filtered through Bowman's capsule, from the glomerulus into the tubules, 60 to 100 liters of fluid. Yes, the appalling total of 15 to 25 gallons. All of the water but about one and one-half liters, which pass on into the bladder, is reabsorbed in its course along the tubules. The water in our body is indeed a vehicle for transportation, and it does keep moving.

A sixth function of water in our bodies is the part it contributes to ferment action. The enzymes, amylase, protease, and lipase break down polysaccharides, protein molecules, and fat, by a hydrolytic process. Water is driven into the large molecules like a wedge, breaking off smaller molecules until the whole is reduced to an available form to be absorbed and utilized by the tissues. Ferments as a class act better in dilution. More water will result in more end products by ferment action.

In the seventh place, we find that water is essential to some of the special senses. Substances to be tasted must be in solution. Again, the waves of sound are transmitted to the organ of Corti in the cochlea by water. The aqueous humor of the eyeball is essential to sight.

Last, water is the medium that is used in regulating body temperature. By virtue of water's high-specific and high-latent heat, it is the ideal stabilizer of the body temperature. It takes more heat to raise one gram of water one degree than any other substance. The billions of cells in our body live an aquatic life. They are surrounded by water. Like the water jacket of an automobile, this water absorbs the heat of oxidation from the cells and prevents their self-destruction. In the liver and the muscles, where the greatest amount of heat is produced, it is taken up by water which keeps on moving throughout the circulatory system, equalizing the temperature in all parts. If the temperature is too high, the latent heat of vaporization is liberated on the surface of the body through the sweat glands.

It was stated that 75 per cent of the body's weight is contributed by water. In studying the location of this water, it has been determined that approximately one-half of the body water is within the cells. Here it is combined with colloidal material. This intracellular water is known as "bound" or "living" water. It it rich in potassium phosphates.

Peters, in his exhaustive review, *Body Water,* states that more than 20 per cent of the body weight is intercellular or interstitial water. This is free water. It is rich in sodium chloride and poor in colloids. This water is the great buffer against changes in the cell on the one hand and the blood plasma on the other. This intercellular water maintains an osmotic equilibrium with the potassium containing intracellular water.

The third place water is found is in the blood vessels, in plasma, and also in the serous cavities. Ten per cent of body weight is contributed by these fluids. Plasma is rich in protein molecules. These exert an osmotic pull which tends to retain water within the vessels. It is important to remember that the body water is within the cells, between the cells, in the vascular system, and in serous cavities.

To maintain the normal amount of body water, an adult must have from 3 to 5 liters of water per day. The available sources of this water are: First, from water ingested as beverages, or water as such. A minimum of 1½ liters must be had in this form. Second, there is the water contained in so-called solid or semisolid foods. This is called "hidden water," and furnishes a little less than one-half the required amount. Third, there is formed in the tissues, water of oxidation. Any substance containing hydrogen liberates water on oxidation. Water and carbon dioxide are the end products of combustion. It has been found that 100 grams of fat, when oxidized, will yield 105 grams of water, 100 grams of protein yield 40 grams of water, and 100 grams of carbohydrate liberate 55 grams of water on oxidation. It is from these sources that the tissues obtain this much needed substance. Water comes first in quantitative importance among the biochemical compounds needed for the metabolic activity

within the body. It has been said that water content of mammalian tissue is proportional to its physiologic activity, and is inversely proportional to the fat content. Water content of the body decreases with age.

There is maintained in the body a water balance between the total water obtained from all sources and that eliminated. The channels by which water leaves the body are: First, the kidneys, some 1,500 c.c. escaping this way. The skin and lungs together remove another 1½ liters of water, while the alimentary tract disposes of 150 cubic centimeters.

There is a compensatory mechanism between these channels for the elimination of water from the body. This is especially true in the case of the skin and the kidneys. An overactivity of one will lessen the output of the other.

Any pronounced alteration in the quantity of body water, in its chemical composition, or in a pronounced shift in its location in the tissues, is followed by a disturbed cellular metabolism. This presents the problem of the physiology of water in disease.

References to the literature for this and the following article are combined and appear on page 457.

THE PHYSIOLOGY OF WATER IN DISEASE

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MARKED variations from normal in the amount, the distribution, or the contents of the water in the tissues indicate a pathologic condition. One of the most serious disturbances of this water balance is that of dehydration. When the loss of water from the body exceeds the combined intake of water and that produced by tissue metabolism, a condition of negative water balance or dehydration results. This is a very serious complication, and presents one of the major problems in the practice of medicine and oral and general surgery. If not corrected early, the patient passes into a state of shock. A loss of 10 per cent of the body water can occur without marked changes in blood plasma, but as the loss advances to 20 per cent, the patient develops profound shock.

The amount of this water depletion depends upon the condition causing it. First, there are the cases of insufficient ingestion; they just do not drink enough water. It has been noted that 3 to 5 liters is the daily requirement for an adult. One-half of this must be taken as beverages. Mental cases often refuse to drink, and become early victims of dehydration. Cases of coma or unconsciousness must be given water freely to avoid this additional complication.

A second cause is that of vomiting. The secretions of the gastrointestinal tract are normally reabsorbed. In cases of severe vomiting this does not occur. These secretions are formed from the water and the electrolytes of the blood plasma, and their loss upsets the fluid balance of the body. Not only are these secretions lost, but a vomiting patient fails to ingest water and thereby is doubly deprived of fluid.

Then there is dehydration caused by diarrhea. This is especially common in infants. Diarrhea can lead to a loss of 20 per cent of the body weight in water in a period of forty-eight hours, with a rapid fatal termination. In severe cases of this type, both intracellular and extracellular fluids are lost. Both sodium of interstitial fluid and potassium of intracellular fluid as well as water need replacement.

Hemorrhage is another cause of dehydration. This is a problem faced by all dentists and physicians who are guardians of their patient's health. The important disturbance in these cases is the loss in plasma volume. The loss of plasma proteins is extremely serious, for they are the most important factors in maintaining fluid in the vascular system. It has been shown that about one-half of the loss by hemorrhage is replaced from the interstitial water in the skin and muscles.

Fifth, body water is depleted by exposure to high temperatures. The greatest factor in heat stroke is the disturbed crystalloid content of the

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tissue resulting from loss of body salt rather than the loss of water per se. It has been found that the collapse during artificial fever treatment can be greatly reduced by careful replacement of water and salts which are lost in sweat. This collapse closely resembles the shock of dehydration.

Edema is another common disturbance of water distribution in the body. It results when an abnormal amount of fluid accumulates in the intercellular spaces. The most common location for this is in the subcutaneous tissues. This intercellular area in the body has been likened to a swamp. Fluid passes into or out of this extensive reservoir of water. In the case of edema, the swamp is overfilled. Swelling, distention of the tissues results from failure to drain off the fluid as fast as it accumulates. This overhydration of the tissue interferes with normal cellular activity. To understand the shift of fluid which brings about edema, it might be well to visualize again the broad capillary bed of the circulatory system, and to remember that the blood pressure at the arterial end of the capillaries is approximately 35 mm. Hg. At the venous end it is 15 to 20 mm. Hg. Opposed to this is the osmotic pull of plasma proteins which is equivalent to 20 to 25 mm. Hg. The result is that the outward filtration thrust at the arterial end of the capillaries will force fluid out into the tissue spaces, whereas the pull of plasma proteins will draw it into the venous end of the capillaries. All of the fluid which escapes at the arterial side of the capillary bed is not returned directly to the venous end of the capillaries, but some finds its way from the intercellular spaces into lymphatic capillaries which returns it to the subclavian veins via the thoracic duct or right lymphatic duct. Many things can disturb this orderly exchange between intravascular and intercellular fluids. In discussing his thorough study of this subject, Cecil Drinker states, "The consequences of abnormal capillary leakage have not been properly appreciated."

One can think of the waves which spread out on the beach and then recede again. If the sand is fine and closely packed, the water will all return between waves, but if the sand is coarse, much seeps through to return by way of the deeper channels. So it is with the circulation. Each stroke of the heart forces the blood over a broad capillary bed. If the endothelial cells of the capillary walls permit leakage, a greater quantity of fluid will escape into the tissues. This is what happens in the great many cases of toxemia with edema. The toxic products of infectious organisms and of tissue destruction act upon the capillary walls decreasing the barrier to the passage of water. Histamine causes capillary dilatation and increases permeability, producing local edema. Urticaris and angioneurotic edema are examples of localized edema which result from increased permeability in restricted areas of the capillary bed.

Any obstruction to the lymphatics will back up the lymph drained from the tissue spaces distal to the obstruction, with edema resulting.

The edema which develops in decompensating heart disease is not due entirely to the general circulatory impairment, but to a great extent it is caused by the fact that sodium chloride is retained in the tissues, holding water because of passive congestion in the kidneys. This venous back pressure in the kidneys prevents the elimination of sufficient fluids and salts. To administer saline solu-

tion to such patients would surely increase the possibility of congestive heart failure.

In the last few years, it has become increasingly evident that a decrease in plasma proteins will produce edema. When the total protein content of plasma drops below the normal of 8 per cent, the inward pull of the plasma ceases to balance the outward filtration thrust of blood pressure. The pressure gradient tends to be reversed at the venous end of the capillary bed. The swamp becomes overfilled, and edema, dropsy, or anasarca develops. This is undoubtedly what happens in nutritional edema, famine edema, or war dropsy. In man's diet, protein is the most expensive as well as the most difficult to obtain of all the food requirements. This is the case in great crises, such as economic depressions, wars, famines. If the diet is insufficient in this principle, the plasma is slowly depleted and edema follows. The edema of nephritis resembles nutritional edema in that it too results from a decrease in plasma protein. In nephritis, the nephron has been injured. The kidneys are unable to hold back the serum albumins and serum globulins of plasma, and they pass through with the urine. Today a case of Bright's disease is given protein for replacement; it is not withheld from the patient's diet as formerly. Again, saline or glucose solution administered to these patients would simply increase the passage of water from the vessels into the tissues.

It is desired to offer the suggestion that dehydration and edema in varying degrees are the two most common disturbances of water balance encountered by the clinician. Further, that in most cases these conditions are too often allowed to reach an advanced stage before they are recognized and proper corrective measures are administered. One of the simplest methods of early determination of these conditions is to measure the specific gravity of the patient's plasma.

There are many other less common, yet very definite disturbances of water metabolism which are encountered. Peters says, "The hormone of the posterior lobe of the pituitary gland seems to contribute to the kidney some power to reabsorb water in the tubules." McQuarrie, in his article in the *Annual Review of Physiology* of 1945, supports this view. Van Dyke concludes "that the reabsorption of water in the kidney tubules after glomerular filtration is due to a diuresis-inhibiting or vaso-pressor hormone secreted by the pars-nervosa of the hypophysis." There is no doubt that one of the main agencies involved in the regulation of renal activity and of water balance is derived from the posterior lobe of the pituitary. Many other men in this field of research have found similar evidence. This is not a great deal unlike Cannon's theory of the adrenal control of the blood sugar level.

Diabetes insipidus presents a definite syndrome, the principal features of which are extreme polyuria and profound polydipsia. The urinary output may reach many gallons per day, and the thirst and craving for water becomes extreme. Without this hormone from the pituitary the kidney cannot hold back water in the tubules, and a great quantity of dilute urine of low specific gravity results. This condition is greatly relieved by injections of posterior pituitary extract.

The adrenal cortex is another of the endocrine glands which influence the water and mineral metabolism of the body. Insufficiency of this gland is accompanied by a decided loss of sodium. This in turn reduces the interstitial fluid of the tissues. Potassium concentration is increased. In the extracellular fluid sodium is the main cation, within the cells it is potassium. Normally these two fluids are in osmotic equilibrium, separated only by a semipermeable membrane through which water and electrolytes readily pass. This loss of sodium disturbs the normal equilibrium of water in the tissues. It is accompanied by an increased elimination of water, and dehydration develops. This condition can be greatly benefited by an increased sodium intake and a reduced potassium consumption. Injections of cortical extract correct this imbalance of the base content of these fluids.

Epileptic attacks are accompanied by an abnormal disposition of body water. McQuarrie makes the statement that "Superhydration of nervous tissue may be a factor in producing epileptic seizures." He also suggests the restriction of fluids in these cases. It does seem, from recent studies, that an increased concentration of water in the body, and especially in the brain, is a predisposing factor in producing convulsions. McQuarrie made the further observation that convulsions were more frequent when an excess of water was retained by injecting the antidiuretic principle of the pituitary gland. Also, he observed that seizures were less frequent during fasting or on the ketogenic diet of high fat, low carbohydrate. Changes of the normal acid base balance to the alkaline side were followed by more frequent seizures.

Fevers in general present an interesting physiologic reaction in relation to body water. It would seem logical that the increased temperature of fever must be due either to an increased production of heat or to some interference in the heat loss. Kraus found that the greatest increase in heat production during high fevers was only 20 per cent above normal. In comparison to this, it is a well-known fact that muscular work may increase heat production from 100 to 300 per cent, with only slight rise in body temperature. The conclusion would be then, that fevers are not due to a great increase in heat production. Investigation seems to indicate that the rise in temperature is due to an interference with the body's heat-dissipating system. In fevers there is paleness of the skin, constriction of the peripheral blood vessels, and absence of sweating. It is a case of the failure of the water-cooling system. The water cannot get to the surface to get rid of its latent heat. If the water pump on the automobile fails, the radiator cannot serve its purpose. Start the fever patients to sweating, as happens at the crisis of some fevers, and the temperature will drop.

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THE INVESTIGATION OF PSYCHOSOMATIC MECHANISMS IN DENTAL PATIENTS

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YOU are all familiar with the findings of psychosomatic medicine that many human diseases may have immediate or remote causes in emotional disturbances, that pathologic processes in various tissues or organs of the body may affect neuropsychic equilibrium, and that "psyche" and "soma," "mental" and "physical," are merely useful verbal fictions with which to designate and classify certain aspects of pathologic processes which are essentially indivisible. Psychosomatic medicine might better be called Human medicine. It ranges beyond the limits of morphologic and physiologic studies of human pathology to include investigations of the patient as a person. For any patient is a member of human society and therefore subject to the pressures exerted by custom and tradition from his racial past, to the stresses and strains of the social conditions of his times, and to his hopes and fears about future social relationships. Human beings are, within limits, more influenced for health or disease by the values they have than by the actual facts of their physical surroundings.

The investigation of psychosomatic mechanisms, then, is based upon these assumptions. It is simply a matter of extending the range of ordinary clinical investigation. In the old way one obtained an accurate history of the onset and course of an illness and a background history of familial and past personal health. But the patient as a person was studied very little if at all. This was especially true if the illness appeared to be "organic," that is, involving recognizable lesions of some sort. For the Virchow influence in pathology blocked the way to our contemporary knowledge that macroscopic lesions are preceded by microscopic changes and these by submicroscopic, physicochemical alterations which might originate as much from nervous disturbances of homeostasis as from biophysical causes. When history taking is extended beyond the range of biophysical happenings in a patient's life to include relevant features of his psychosocial history, it frequently becomes strikingly evident that the patient's thoughts and feelings have much to do with the origin and course of his illness.

The technique of obtaining such an extended, psychosomatic history involves certain problems of physician-patient relationship. Either or both may be reluctant to discuss intimate matters of personal history. This is

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especially true for dentists and dental patients who act on the assumption that their professional relationship is restricted to the mechanical treatment of exclusively dental lesions. You, as members of this seminar in dental medicine, have gone far beyond the anatomic features of a fictional "oral cavity" in your search for pathogenetic factors, and you have more knowledge of the psychosomatic mechanisms than many physicians. But your patients are not so enlightened and may even be defensive when you seemingly take their lives apart in your search for the causes of their dental problems. They may be surprised and even resentful at your nutritional investigations, let alone any well-meaning intrusion into more personal aspects of their living habits. It is essential, therefore, to win the patient's trust and cooperation before starting such an exploration of his personal life; and it is necessary to develop a gentle, efficient technique of investigation which will enable you quickly to obtain the data needed for psychosomatic diagnosis.

The dentist is, despite his lack of formal psychiatric training, in a better position to obtain confidences than he may believe. In the main, people like and trust their dentists, though they may also fear them. This is evident by their gossiping about them, their possessive pride as they speak of "my dentist," and even the ubiquitous joking about dentists. This probably stems from the fact that the good and bad characteristics of our oral apparatus symbolize some very deep evaluations concerning the security feelings of love and feeding and the anxious states connected with defense and aggression. To have one's dental apparatus manipulated is bound to evoke such deep emotional resonances. If the dentist is aware that he unavoidably stirs up infantile reactions of love and fear, he is in a unique position to make some diagnostic observations. Therefore, he should cultivate attitudes toward his patients of warm friendliness tempered with benevolent neutrality and, by his own silent example, should reassure the patient of his discretion about confidences. He should be a good listener, saying little except to direct the patient's conversation into productive channels, and in his listening to be attuned to the emotional undercurrents of what is talked about; for the patient's comments about business and politics may reveal as much of a person's emotional life as the frankest confessions.

There are many practical signs to observe. For example, the way in which a person makes and keeps appointments (urgently, tentatively, reluctantly, et cetera) and his tension or ease in the chair. Physiologic signs of tension are well known to you: the dilated pupils, sweating and hyperventilation, dry mouth, and trembling or grasping movements, among others. Look also for the signs of defense against anxiety such as talkativeness and bravado, flippancy and indifference, postponement and rationalization, truculence, and the use of drugs or alcohol. The dental tissues themselves may reveal the destructive effects of nervous sucking or grinding habits, overzealous or neglectful hygiene of neurotic type, and faulty nutritional habits. The patient's attitude toward the reconstruction and the monetary arrangements proposed, should be noted. From session to session, the dentist should watch all the reactions of his patient so that he becomes aware of physiologic and conduct patterns that characterize a given personality.

The actual interrogation, as far as psychosomatic mechanisms are concerned, commences where the ordinary anamnesis leaves off. It is directed, as we have said before, toward an investigation of stresses and strains within the personality, and their relationship, if any, to the pathologic process. Oriented in this way, not only must the interrogation be tactful, to avoid provoking defensiveness, but the questions should be indirect, as far as possible, to obviate positive and negative suggestions. It would be as unwise to ask "Do you worry?" "Are you happily married?" as to ask inferential questions of the type, "Have you stopped beating your wife?" Many patients would be ashamed to admit that they worried and certainly most would deny anger, resentments, or marital doubts even if they were conscious of such reactions. And once the patient has given a purely conventional answer, or a false or misleading one, he has to maintain his fictions with the result that much time and progress in diagnosis and treatment may be wasted.

Actually, as you know from our previous studies, patients are frequently unaware of their desires, angers, and hates for the sufficient reason that such reactions involve their interpersonal relations and their admission could lead to self-recrimination or threats to security. The energy involved in restraining such impulses, and the fears of their consequences produce, of course, cortico-diencephalic imbalances leading to those disturbances of homeostasis which may long precede microscopic or macroscopic lesions. Therefore, the interrogation should at first avoid, or at least postpone, direct questioning in the areas of love life and hostile feelings. The manner in which the patient speaks about himself, his family, his business associates and friends, and the obvious eagerness or evasion he shows as he does so, give valuable data which may later be confirmed by spontaneous admissions as confidence in the physician is established. Patience should be the keynote in psychosomatic investigations.

Questioning should be directed at first to the time-setting of illness, since this logically follows the patient's recital of complaints. The aim is to detect any connections between periods of social stress, such as economic or marital changes, and disease processes. The time of onset and the duration of every period of ill-health which the patient has suffered should, ideally, be ascertained and each item should be placed in the social setting of that period in his life. Here again it is best to be indirect in your questioning, systematizing the information for your record later. For example, having obtained the usual statistical data, such as name, age, marital and economic status, by direct questioning, and having noted that the patient was divorced three years ago, your history of the patient's dental troubles reveals that severe paradentosis set in four years ago and thus coincides with the social problem of separation and divorce. You go farther back in the patient's life and find previous attacks of pyorrhea or other diseases, and even accidents, occuring at times when security was threatened, perhaps by the divorce or death of parents, by economic disaster, by changes of schools or jobs. By this method you build up a picture of the sequence of disease processes (systemic and dental) and the pattern of their relationships to episodes in the patient's social functioning. You will be struck by the concurrence of all types of illness with actual changes

in, or threats to, social status; and when you learn more of the patient's inward life, his thoughts and feelings about others, you will see an even greater correspondence between attitude and illness.

The following chart, adapted from one designed by Dr. Stanley Cobb in his excellent little book entitled *Borderlands of Psychiatry* (Harvard, 1943) is based on actual case material. Its value lies in the time-setting relations it reveals.

TIME-SETTING CHART

NAME: L. F. AGE: 29 YEARS SEX: MALE CASE NUMBER

YEAR	DENTAL DATA	MEDICAL DATA	SOCIAL DATA	AGE
1916		Normal birth	Oldest of six siblings	0
1919	Reverts to thumb-sucking	Temper tantrums	Birth of second child	3
1924	Breaks upper incisors in fight		Unpopular at school due to bellicosity. Father alcoholic	8
1930	Caries	Tonsillectomy and adenoidectomy	Father severely alcoholic and failing in business. Patient working after school	14
1931	Caries	Nasal polyps removed	Envious of boy friend because of his own poverty	15
1932			Joins U. S. Navy to get away from home. Hazed by sailors for "temper." "Controls" temper	16
1936	Grinding of teeth. Caries		Father dies of pneumonia	20
1938	Continued grinding		Leaves Navy	22
1939	Continued grinding and caries	"Stomach trouble"	Resented sending money to mother	23
1940	Continued grinding and caries	"Stomach trouble"	Resented sending money to mother	24
1941	Continued grinding and caries	Headaches	Stopped sending money home	25
1942	Continued grinding and caries	Headaches increase nasal congestion	Marries an older woman and insists she give up work to keep house	26
1943	Continued grinding and caries	Headaches worse. Nasal operation	Great difficulty controlling temper. Difficulty with foreman at machine shop	27
1944	Continued grinding and caries	Headaches severe	Friction with wife and foreman	28
1945	Continued grinding and caries	Headaches improved	Re-enlists in Navy	29

Useful as such a scheme is, it reveals little about the underlying attitudes of a person which may be playing a role in the production and maintenance of illness. It shows only the superficial correlations, not the meaning of the patient's reactions, although your intuition will certainly enable you to make some shrewd guesses from such data. More information is necessary—information about the patient as a member of a contemporary society meeting certain established forms of friendship, occupation, and love relationships. What of his capacities to satisfy his needs in terms of social responsibility? Does he properly evaluate his potentialities and limitations? Is he flexible in his adjustments due to a healthy sense of realities, or is he rigid due to a neurotic shell of defensive reactions arising from his distorted views of life? These, and related questions, must be answered for a complete diagnosis, and although they may be organized and recorded according to the following scheme,

the actual interrogation of the patient should be spontaneous and informal rather than systematic.

I. Family history and social background:
  A. Grandparents.
  B. Parents.
II. Intrafamilial relationships:
  A. With parents.
  B. With siblings.
III. Individual development:
  A. Infancy.
  B. Childhood.
  C. Adolescence.
  D. Problem behavior.
IV. Maturity:
  A. Friendships and interests.
  B. Occupational history.
  C. Sexual history.
V. Psychological data:
  A. Earliest memories.
  B. Dreams.
  C. Fantasies, wishes, and fears.
  D. Moods.
  E. Self-estimate.
VI. Psychological tests.
VII. Social investigation.
VIII. Diagnostic summary.

Family history becomes more than a search for familial disease tendencies. We need to know something of the national, religious, and economic origins of the parents and even the grandparents. Such data will reveal cultural clashes greatly affecting the patient during his childhood. Consider differences of economic station, religion, education, and the like between parents and between generations. Do they not profoundly influence the values a growing child selects? Compare the relentless drives for power prestige and possession that appear in the many people of lowly origin with the less tense attitudes of personalities raised in greater social security. Patients will reveal much of their own attitudes as they tell you of their grandparents and parents: they may show pride or shame, a keen awareness or apathetic ignorance of their historical backgrounds.

Having learned something of the biologic and cultural heritage, we must study the actual relationships within the family. This will involve such factual data as ages, health, religion and occupations of parents, death separations and divorces, step or foster parentage, the number, ages, and sex of siblings (brothers and sisters) and even their health, occupation, and marital status. An important orientation should be followed here: this is to date all family events such as births, marriages, illnesses, deaths, separations, etc., by the year they occurred in the patient's life. We wish to find what changes in family relationships, such as deaths and births, may have influenced his feelings of occupying a secure place in the family constellation. The absence or separation

of parents, the introduction of step or foster parents, the rivalries between brothers and sisters play a large role in the first years of life in determining future social relationships. Seemingly the structure of our relationships with marital partners, friends, and occupational associates of later life is but a magnified projection on the screen of adult society of the images we formed of ourselves and others in the family circle of childhood.

A developmental history of the individual for psychosomatic purposes involves more than a record of mere "physical" events such as teething or illnesses. As you know, the nursing and feeding history, the methods and results of toilet training, the presence of, and methods of dealing with, habits like thumb-sucking and masturbation involve social relationships. The somatic organs and mechanisms involved in nutrition, warmth, and cleanliness, elimination, and sexual pleasure become the vectors of interpersonal values. Thus the oral zone, the upper intestinal tract, the sense organs, the skin and muscles associate the search for, and attainment of, food and warmth with generous or rejecting human relationships. Similarly, the lower intestinal tract, the anal zone and the contiguous urinogenital organs are conditioned by strong interpersonal experiences involving cooperation and love or discipline and fear. Physiologic functions become psychophysiologic; our feelings toward others, past or present, are frequently expressed by a "body language" in which organic changes, reversible or irreversible, express nonverbal evaluations.

It is important, therefore, to record these aspects of developmental history carefully. To them may be added the play life and school life of childhood, the onset and problems of adolescence, the attainment of maturity and independence. A biography of the patient's life cannot be too complete; yet it is only the stresses and strains of the social adjustment that really concern us. We continually ask ourselves the questions: How has the patient dealt with the problem of social adaptation? Does ill-health reflect some interpersonal strain? Dividing social relationships into three interconnected sectors of sex, society, and subsistence, as it has been put alliteratively, is a convenient guide for interrogation and later evaluation of historical material, since we have certain norms of conduct in mind for the age, sex, and education of every person. Is the patient above or below standard in any of these fields? Does success in one, such as occupation, compensate for deficiency or failure in another, such as marriage? Or is he below par in all three? Is his participation in life wide or restricted? Is this consonant with his age, position, and abilities?

A history of the patient's choice of, and ability to make, friends outside his family and business life will indicate seclusive or gregarious trends which may reflect deep feelings of inadequacy, needs for power and attention, or a healthy sense of interdependence. Clubs, church connections, and informal social connections should be noted, and along with them the patient's attitudes toward such affiliations.

The occupational record is always revealing. It shows us the ambitious drive and abilities of a patient and a great deal about his capacities to work with others. Much psychosomatic illness will be related to stresses in the occupational situation, such as failure to be promoted or recognized, promotion

to responsibilities the patient fears, irritations from working associates, and the like. A careful search for satisfactions and dissatisfactions in the economic and professional relationships should be made. Attitudes toward work and money have very deep roots in a personality because of their importance to social security.

Investigation of the patient's sexual history is best left until a time when the patient's confidence in you is stronger. Great tact, yet a freedom from personal embarrassment in asking questions will be necessary. Premarital, marital, and extramarital problems and the patient's reactions to them should be noted. Very frequently deep anxieties will be found about masturbation, impotence, and frigidity, and the so-called "perversions." While these are usually matters for expert investigation and treatment, there is no question that tolerance and reassurance on the part of the listener will do much to relieve the patient of anxious burdens. False-to-fact knowledge, guilt and anxiety about sexual problems are among the most potent sources of tension in our lives today and many psychosomatic reactions can be the consequence.

When the more or less "objective" data about social background and individual development have been collected, usually without much reluctance on the patient's part, we come to the investigation of more subtle aspects of the patient's functioning—the highly "subjective" psychological sphere. The interpretation of material from the psychic life requires specialized training and experience but there is no reason why the general practitioner should not collect some psychological data and, as his experience widens, begin to see its significance. One should inquire after the patient's earliest recollections, his early school memories, his childhood dreams and fantasies. Then we should obtain samples of recent and recurrent dreams, expressions of fears, wishes, and beliefs. Finally the patient's self-evaluation—his awareness of his own happiness or depression, his own worth as a person—should be elicited. Statements about these matters should be recorded as nearly verbatim as possible since the very language used may provide valuable clues to the diagnostic problem.

If the services of a competent clinical psychologist are available, ask him to administer appropriate tests such as the Wechsler-Bellevue "intelligence" test for adults and the Minnesota Inventory for personality traits. These are so-called "objective" tests, depending on the patient answering direct questions, solving standardized problems, etc. Then there are "projective" tests; for example, the Rorschach ink blots and the Murray pictures, which study the patient's imaginative and interpretative capacities. In the hands of an expert they reveal a great deal of diagnostic importance.

An invaluable supplement to the study and interrogation of a patient in the office is a visit to the patient's home to see him in his natural environment and to observe the family (or work) relationships directly. This is best carried out by a trained social worker. In our offices we are often presented with a highly edited account and dressed-up appearance by the patient. This is one of the costs of modern specialization. The country doctor visited the home, saw the relatives, knew the friends and fellow workmen, and thereby could

check objectively the patient's subjective evaluation. He saw the total life situation. As specialists, we need to supplement our eyes and ears by the observations of a trained social investigator. This is particularly true for children's problems where friction in the nursery, at mealtimes, and at school may never be elicited by direct questioning. The employment of trained social workers, along with dental assistants and hygienists, would not only extend the range of investigation but also the therapeutic influence to the home. Social workers are specially trained to investigate and record the data of psychosomatic disorders and could free the dentist of this responsibility. Furthermore, many social workers can carry out varieties of psychotherapy. It is a good practice, when diagnostic material from various sources is collected, to summarize it. It should be a brief paragraph integrating the significant stresses, their origins, and their consequences in a given patient. It should be a word portrait of the total situation. "Diagnosis," said Galen, "is the awareness of all things present" in a case. It is not mere labeling. Only from such an "awareness of all things present" will we be able to comprehend psychosomatic illness.

THE EFFECT OF HEAT-PROCESSED FOODS AND METABOLIZED VITAMIN D MILK ON THE DENTOFACIAL STRUCTURES OF EXPERIMENTAL ANIMALS

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WE PERFORMED a feeding experiment with cats in our laboratory a few years ago to determine the effect of heat-treated foods upon growth and development. This experiment stemmed from the fact that we suffered steady mortality among the cats on which we were performing adrenalectomies for the purpose of standardizing adrenal cortical material. We were feeding these animals the meat scraps from the Sanatorium, together with raw milk and cod-liver oil. The cats were poor operative risks although our technique was good. In time, more cats had been given to us than we were able to feed on the scraps from the Sanatorium. We placed an order for raw-meat scraps at the market where the Sanatorium meats were bought; these scraps included muscle, bone, and viscera. This raw meat was fed each day to the same group of cats. Within a very short time the cats in those pens survived the operations, the unoperated cats appeared to be in better health, and the kittens born were vigorous. The contrast in apparent health between the cats in the pens fed on raw-meat scraps and those fed on the cooked-meat scraps was so startling that we decided to do a feeding experiment.[1-3]

The cats were kept in open-air pens with a yard 4 feet wide, 7 feet high, and 12 feet long, one end having a roof, and a floor with bedding.

A routine procedure was followed with all cats. They were weighed, numbered, and described. In the case of donated cats, all possible information was obtained from donors regarding the history of development and the type of food the cat had received before being placed in our pens. Clinical notes, including weights, were kept. Each kitten was described and weighed on the day of birth, giving parentage, behavior of the mother during the birth, and other pertinent data, and clinical notes continued on all viable kittens. In the case of death an autopsy was performed, cataloguing gross and microscopic findings. Calcium and phosphorus determinations were done on the femur. At the close of the experiment, which ran for ten years, all remaining animals were autopsied.

In the first series of experiments, one group of cats was fed a diet of two-thirds raw meat, one-third raw milk, and cod-liver oil. The second group was fed a diet of two-thirds cooked meat, one-third raw milk, and cod-liver oil. Within the ten-year period, approximately nine hundred cats were studied,

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upon nearly 600 of whom we have complete records. The amount of data accumulated is large. In this paper only the general résumé of predominating findings is presented.

The cats receiving raw meat and raw milk (Figs. 1 and 2) reproduced in homogeneity from one generation to the next. Abortion was uncommon, litters averaged five, and the mother cats nursed their young in a normal manner. The cats in these pens had good resistance to vermin, infections, and parasites. They possessed excellent equilibrium; they behaved in a predictable manner. Their organic development was complete and functioned normally.



Fig. 1.—Adult female cat, one year old, on raw meat and milk. First litter in pens, 1936. Four kittens, 4 days old. Note full development and act vity of kittens. Note smoothness and luster of fur. Inactive kitten second from left is kitten of cat 524.



Fig. 2.—Four kittens of cat in Fig. 1, 1936 litter, 4 days old, and one living kitten (second from left) of cat 524. Note superior facial and bodily development of the raw-meat kittens.

Cats receiving the cooked-meat scraps (Fig. 3) reproduced a heterogeneous strain of kittens, each kitten of the litter being different in skeletal pattern. Abortion in these cats was common, running about 25 per cent in the first generation to about 70 per cent in the second generation. Deliveries were in general difficult, many cats dying in labor. Mortality rates of the kittens were high, frequently due to the failure of the mother to lactate (Fig. 4). The

kittens were often too frail to nurse. At times the mother would steadily decline in health following the birth of the kittens, dying from some obscure tissue exhaustion about three months after delivery. Others experienced increasing difficulty with subsequent pregnancies. Some failed to become pregnant. For all breeding purposes, except special studies not reported here, we used a normal, raw-meat-fed male of proved fecundity, thus eliminating the possibility of male sterility.



Fig. 3.—Adult female cat 524. On cooked meat and raw milk for six months and during pregnancy. 1936 litter. Note dull eyes, poor fur. Three kittens, 4 days old, dead. Living kitten shown on Figs. 1 and 2.

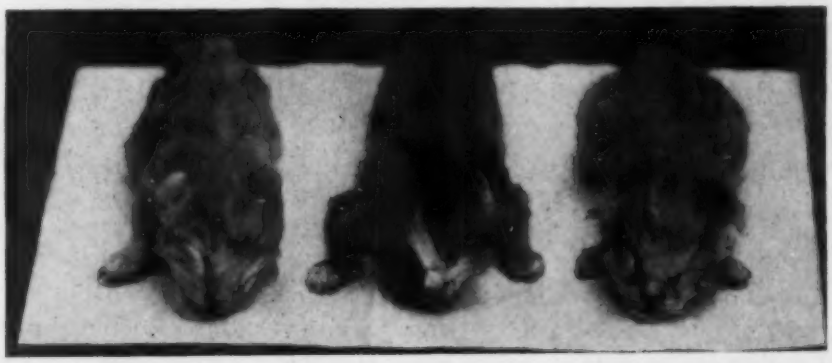


Fig. 4.— Three dead kittens, 4 days old, of cat 524, 1936 litter. Note poor fur and general development.

Cooked-meat-fed cats were irritable. The females were dangerous to handle, occasionally viciously biting the keeper. The males were more docile, often to the point of being unaggressive. Sex interest was slack or perverted. Vermin and intestinal parasites abounded. Skin lesions and allergies were frequent, being progressively worse from one generation to the next. Pneumonia and empyema were among the principal causes of natural death among the adult cats. Diarrhea, followed by pneumonia, took a heavy toll of the kit-

tens. Osteomyelitis was also both common and often fatal. Cardiac lesions, some ascertained clinically during life, were frequent. Hyperopia and myopia, thyroid disease, nephritis, hepatitis, orchitis, oophoritis, paralysis, meningitis, cystitis, arthritis, and many other degenerative lesions familiar in human medicine were observed.

Of the cats maintained entirely on the cooked-meat diet, with raw milk, the kittens of the third generation (Figs. 5 and 6) were so degenerated that none of them survived the sixth month of life, thereby terminating the strain.

One group of cats was fed first on raw meat, then placed on a cooked-meat diet for six months, and then returned to a raw-meat diet. When females of this group became pregnant, their kittens suffered some of the afore-mentioned stigmas, although the females themselves appeared to be in good health. Their succeeding litters would show irregularities that tended to lessen in intensity for the first two or three years, and then to increase again. The generations following improved as long as they received the raw-meat diet. Their resistance to disease, greatly diminished when the cooked-meat diet was administered, gradually improved upon their return to the raw-meat diet. They would partially maintain their skeletal form, but their calcification would continue to diminish (Figs. 7 and 8). Their reproductive efficiency would be injured from the standpoint of the size and vitality of their kittens, and was particularly noticeable in the failure of the subsequent litters to conform to a homogeneous pattern.

Cats of the first and second generation cooked-meat-fed groups were returned to a raw-meat diet. These were classified as "regenerating" animals of the first and second order, first, second, third, and fourth generations.

It apparently requires four generations for either order to regenerate. However, because of lack of reproductive efficiency, it is only the occasional animal that returns to the normal homogeneous pattern noted before deficiency was imposed on the strain of cats. Improvement in resistance to disease is noted in the second generation regenerating cat. Allergic manifestations persist. Reproduction is erratic. In the third generation regenerating animal, skeletal and soft tissue changes are still noticeable to a lesser degree. In the fourth generation, the occasional animal appears to have returned to normal skeletal and tissue form.

At autopsy, cooked-meat-fed females frequently presented the picture of ovarian atrophy and uterine congestion, whereas the males often showed failure in the development of active spermatogenesis. The long bones tended to increase in length in these animals, while diminishing in diameter. It was common to find the hind legs increasing in length over the forelegs. The trabeculation of the bones became coarser and showed evidence of less calcium (Fig. 9). In the third generation of cooked-meat-fed animals, some of the bones became as soft as rubber and a true condition of osteogenesis imperfecta was present (Figs. 10 and 11).

In the cooked-meat-fed animals, visceral volume decreased, which was evident from the size of the thoracic and abdominal cavities. However, the males on the sweetened condensed milk were overly fat and showed distended abdominal cavities.



Fig. 5.—Left to right, kittens 448, 461, and 481. No. 448 is third generation, cooked-meat male, first litter, aged 2 months, 3 weeks. Ca 4.32; P 2.20. No. 461 is regenerating first order, second litter, female, aged 2½ months. Ca 3.51; P 1.79. No. 481 is second generation cooked-meat female, aged 1¾ months. Ca 5.14; P 2.41.

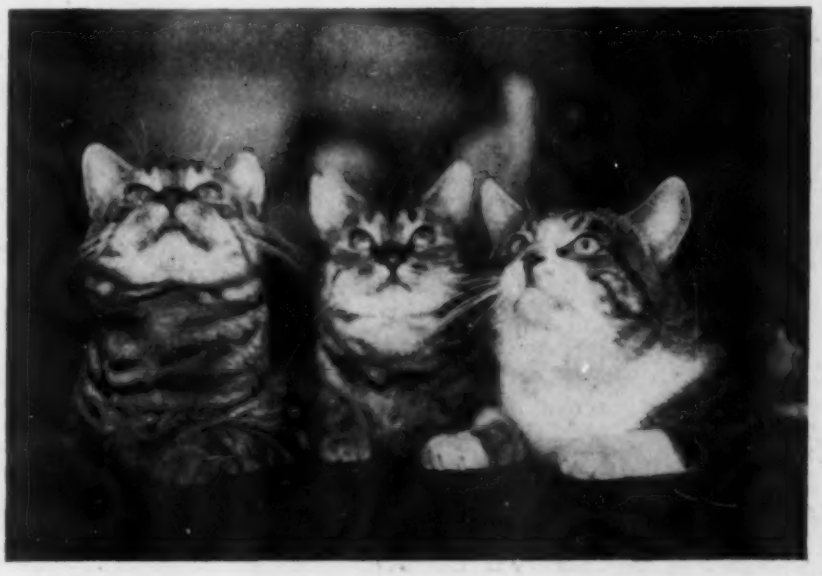


Fig. 6.—Left to right, male kittens 530, 508, and 534, all 18 months of age. No. 530, regenerating, first order, first litter, hypothyroid. Note failure in development of orbital and malar arches. No. 508, second generation, first litter cooked-meat kitten. Note smallness of head, retraction of mandible, and failure in development of orbital and malar arches. No. 534, raw-meat cat.



Fig. 7.—Female cat 538, aged 4 years, 4 months.



Fig. 8.—Female cat 538, as in Fig. 7. Aged 6 years, 3 months. Note continued demineralization of skull and loss of incisors. Opening of bite due to erosion of condyle. Change in position and alignment of canines. Progressive loss of bone from alveolar crests. Note change in shape of muzzle and frontal sinus.

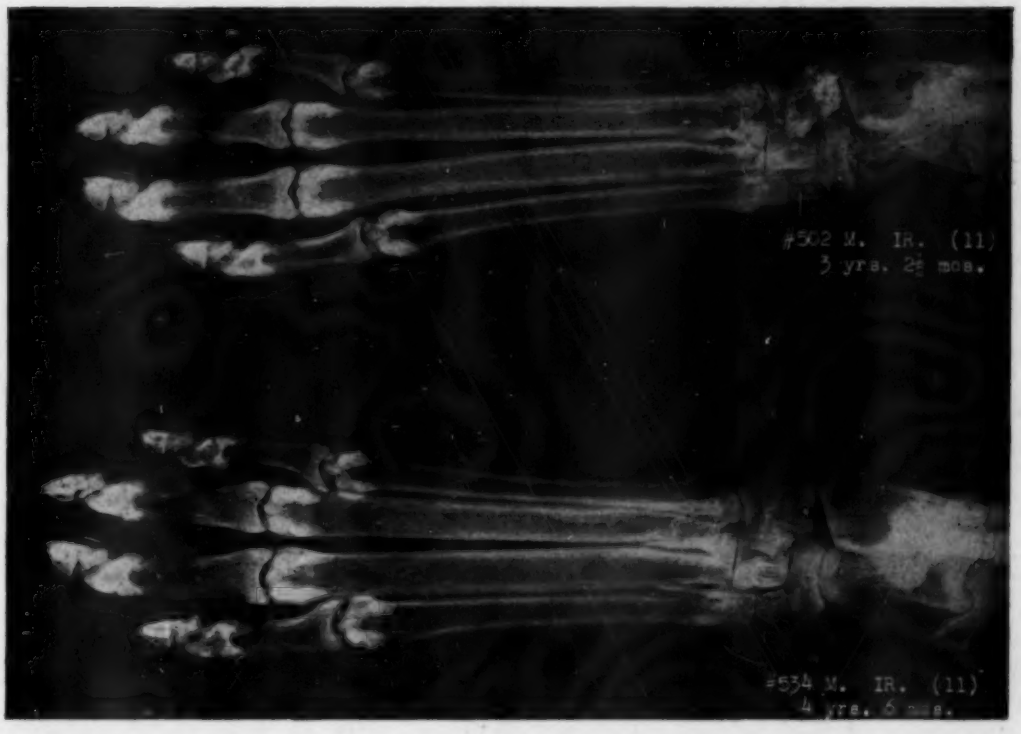


Fig. 9.—X-rays of hind feet of male cats 502 and 534. Cat 502 was the son of male cat 534 and female cat 524. Mother (524) had been subjected to cooked meat diet in 1936. Her kitten (502) was one of the third litter after cat 524 was returned to raw-meat diet. Cat 502 was raised on raw meat. Note coarse trabeculation of the bones of cat 502 when compared with the same structures in the father. Trabecular pattern of mother's foot was slightly coarser than that of the father.



Fig. 10.—Skull of female kitten, aged 3 months, 3 weeks. Regenerating first order, first litter. Ca 3.25; P 1.64 of femur. Note that skull is complete except for orbital arch.



Fig. 11.—Skull of third generation, first litter cooked-meat female kitten, aged 5 months, 3 weeks. Ca 1.86; P 0.639. Note how skull falls apart at suture lines.

Before I describe the dental conditions that obtained in the cats on the raw- and cooked-meat diets, I wish to state briefly that we did three other series of feeding experiments. In these series we used the following kinds of milk: raw milk, raw metabolized vitamin D milk, pasteurized milk, evaporated milk, and sweetened condensed milk (Figs. 12, 13, 14 and 15). Roughly, our results corresponded with those of the previous experiments; animals on raw milk and raw meat reproduced a homogeneous strain, the usual causes of natural death being old age or injuries from fighting.

The male cats fed on metabolized vitamin D milk (from cattle fed irradiated yeast) and raw meat showed osseous disturbances very like those on pasteurized milk, while the female seemed unaffected. However, an interesting circumstance occurred in the males fed on this milk. Young males did not live beyond the second month, and adult males died within ten months. The most notable fact was that there is a tendency for the calcium-phosphorus ratio to become unbalanced, approaching 2.5 to 1, as compared with a normal of 2 to 1. (Tables I, II, and III.) This was accompanied by osseous changes, including the development of rickets in certain of the young animals. As was



Fig. 12.—Internal organs of female cat 533, fed a diet of one-third raw meat and two-thirds raw milk. Note excellent condition of fur and creamy yellow subcutaneous tissue with high vascularity. Moderate heart size. Good liver, firm intestines, and resting uterus. Note that the muscle of the raw-milk-fed animal has a deeper red color and appears more vascular than that of the animals receiving the three heat-processed milks.

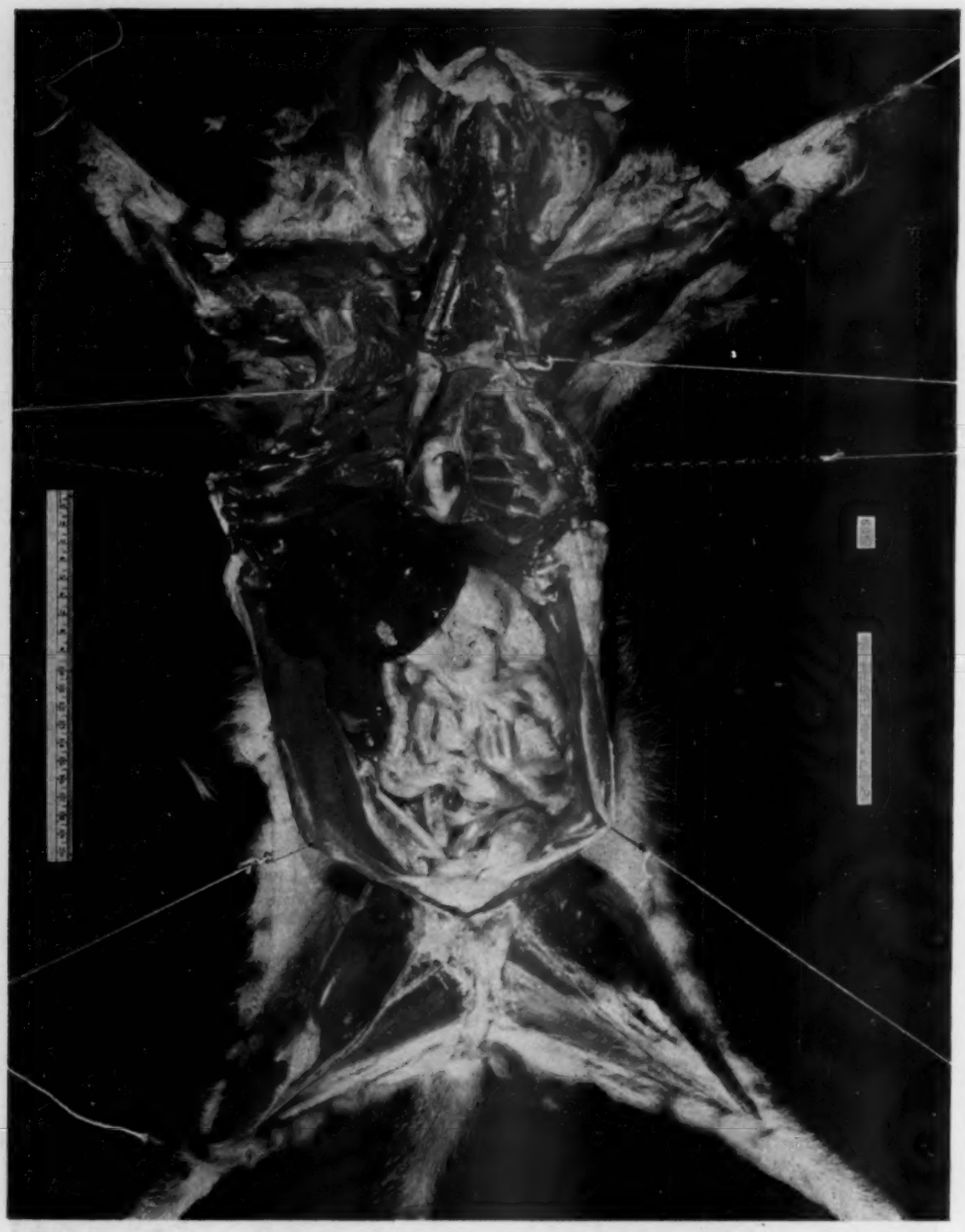


Fig. 13.—Internal organs of female cat 691, fed a diet of one-third raw meat and two-thirds pasteurized milk. Note poor tone of skin and inferior quality of fur. Fair heart. Slight fatty atrophy of the liver. Lack of intestinal tone; moderate distention of uterus. Note the vasomotor disturbance of the skin with a shift from the creamy color of the raw-milk-fed cat to the purplish discoloration of congestion.

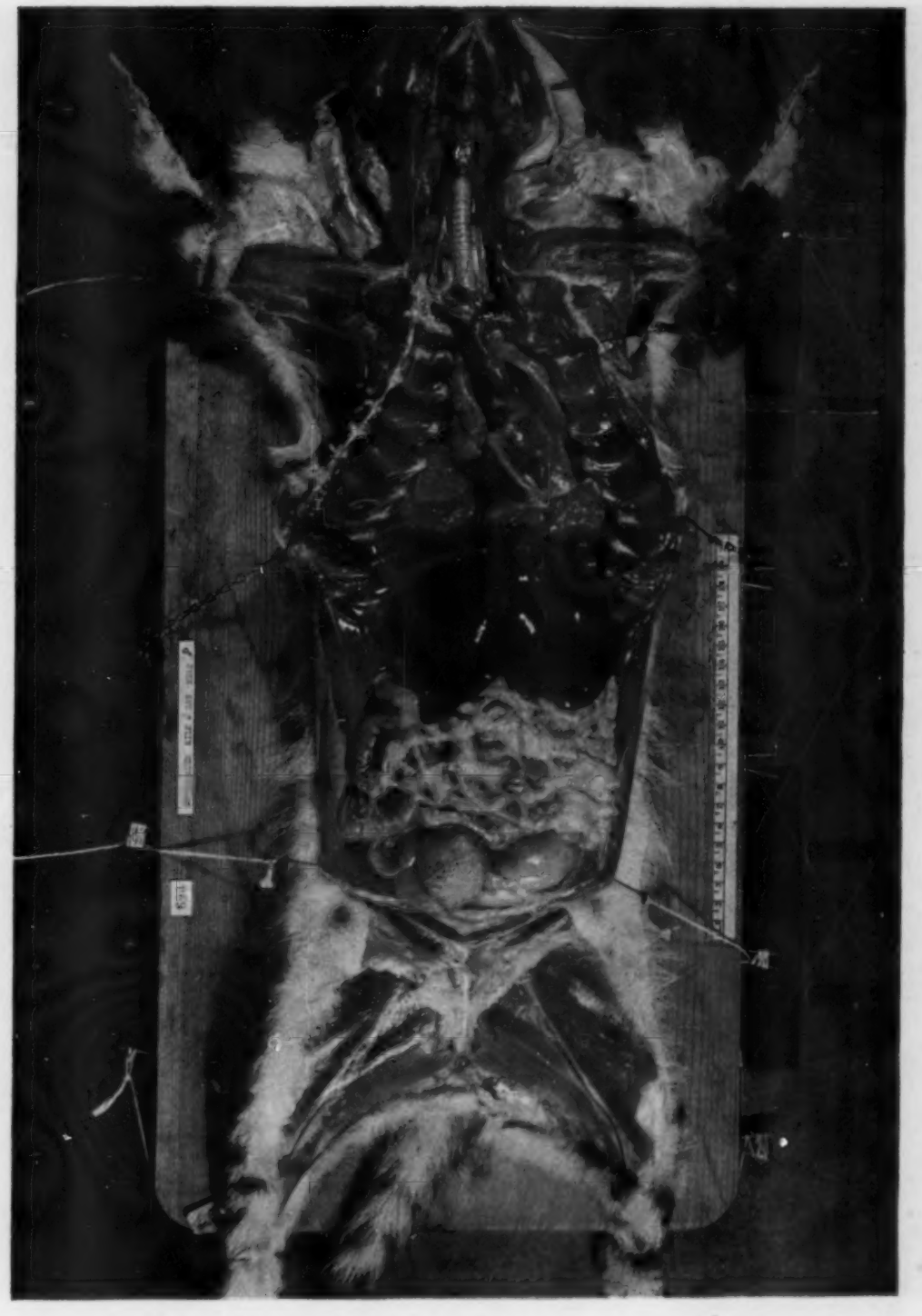


Fig. 14.—Internal organs of female cat 693, fed on evaporated milk and raw meat. Note poor quality of fur and marked purple vasomotor congestion of the skin. Bronze cast to musculature. Failure of lungs to collapse. Slight enlargement of the heart. Chronic passive congestion of the liver; gaseous distention of the small intestine. Genital tract not seen.



Fig. 15.—Internal organs of female cat 506, fed sweetened condensed milk and raw meat. Note extreme coarseness of fur and marked purple vasomotor disturbance of skin. Failure of lungs to collapse. Enlargement of liver and spleen. Chronic passive congestion. Lack of intestinal tone. Marked engorgement of uterus.

TABLE I. LONGEVITY OF IRRADIATED VITAMIN D MILK-FED CATS

(Four litter-mate, well-developed raw-meat male kittens, age 1½ months, were placed on two metabolized vitamin D milks—one from a certified farm where the cattle received green feed, and the other where cattle received dry feed. Note that the dry-feed-fed cattle gave milk which produced rickets when fed to cats, in spite of high amount of vitamin D present. The vitamin D irradiated milk was the sole source of food)

	DIET	RICKETS	FIRST AND LAST WEIGHT	WEIGHT CHANGE	CALCIUM AND PHOSPHORUS RATIO	DATE AND CAUSE OF DEATH
Female 359	Raw meat control	0	605 1,445	+840	(2) Ca 13.27 P 6.72	Chloroformed 6/24/36
Male 360	Green feed, vitamin D milk	0	620 540	− 80	(191) Ca 15.52 P 8.12	Pneumonia 6/16/36
Male 361	Green feed, vitamin D milk	0	580 501	− 79	(202) Ca 15.86 P 7.85	Pneumonia 6/16/36
Male 362	Dry feed, vitamin D milk	Present	640 506	−134	(205) Ca 15.60 P 7.61	Pneumonia 6/15/36
Male 263	Dry feed, vitamin D milk	Present	700 537	−163	(216) Ca 17.71 P 7.27	? Severe Rickets 6/15/36

Litter mates born March 1, 1936.
Experiment started April 15, 1936.
Milk was sole diet.

pointed out by Becks[4] in his experimental work with dogs, natural cod-liver oil did not appear to bother the cat.

The cats fed pasteurized milk as their principal item of diet, and raw meat as a partial diet, showed lessened reproductive efficiency in the females, and some skeletal changes, while the kittens presented deficiencies in development. Cats fed evaporated milk showed even more damage. However, the most marked deficiencies occurred in the sweetened condensed milk fed cats. We believe that the excessive carbohydrate in this milk was responsible for much of this heavy damage.

Later, we made a comparative study of several types of milk on white rats,[5] the general results of which coincided with those found in the cats. Among the findings is the change in trabeculation of the bones, which was well demonstrated in the elbow (Figs. 16*A*, 16*B*, and 16*C*).

Of particular interest are the dental structures of these animals (Figs. 17*A*, 17*B*, 17*C*, 18*A*, 18*B*, 18*C*, 19*A*, 19*B*, and 19*C*). The cats fed on the raw-meat diet from generation to generation maintained a regular, broad face with prominent malar and orbital arches, adequate nasal cavities, broad dental arches and regular dentition. The configuration of the female skull is distinct and different from the male, and each maintained the normal outlines. The membranes were firm and of a good pink color, with no evidence of infection or degenerative change. In older cats, particularly the males that had engaged in much fighting, the incisors had often been lost; but in raw-meat-fed cats of raw-meat-fed ancestry, gingivitis or paradentosis was seldom seen.

TABLE II. MILK EXPERIMENT NUMBER IV

(Experiment to study effect of heat-processed milk in the development and longevity of cats fed one-third enough raw meat to maintain them and two-thirds the amount of food from milk. Note that the animals on the metabolized vitamin D milk diet died within two months, while most litter mates survived the entire experiment. Start of experiment, Dec. 12, 1940; close of experiment, Aug. 13, 1941 [8 months])

NUMBER AND ORDER	AGE AT BEGINNING OF EXPERIMENT	LENGTH OF LIFE AFTER EXPERIMENT BEGAN
Male	*Condensed Milk*	
*788 1 Re L1	9½ months	8 months
749 1 Re L3	7½ months	3 months
757 1 Re L3	7½ months	8 months
Female		
644 Re	Adult	4½ months
646 1 Re L1	1 year 7 months	4½ months
506 Re	2 years 6 months	8 months
Male	*Evaporated Milk*	
789 1 Re L1	9½ months	8 months
750 1 Re L3	7½ months	4½ months
760 1 Re L3	7½ months	8 months
Female		
794 Re	Adult	4½ months
693 1 Re L1	2 years	8 months
678 1 Re L2	1 year 4 months	5¾ months
Male	*Pasteurized Milk*	
790 1 Re L1	9½ months	8 months
752 1 Re L3	7½ months	2 months
761 1 Re L3	7½ months	8 months
Female		
691 Re	2 years	8 months
569 1 Re L1	1 year 8 months	2½ months
532 Re	3 years	8 months
Male	*Metabolized Vitamin D Milk*	
792 1 Re L1	9½ months	1 month 22 days
753 1 Re L3	7½ months	1 month 16 days
763 1 Re L3	7½ months	1 month 11 days
Female		
533 Re	4 years	8 months
697 1 Re L1	1½ years	8 months
694 Re	5½ years	8 months

Males 788, 789, 790, 792—litter mates.

Males 749, 750, 752, 753—litter mates.

Males 757, 760, 761, 763—litter mates.

*Explanation: 1 = First generation; Re = regenerating; L1 = first litter. Example: 1 Re L1 = First generation, regenerating, first litter. It is the first generation raw-meat animal, born to a cat after it had been on a cooked-meat diet.

Adult cats placed on a cooked-meat diet begin to show unhealthy conditions in the mouth within three to six months. A pregnant cat will show the change more quickly. These cats first present gingivitis, then diminished calcium, paradentosis, abscesses, and finally, some shedding of the teeth. The canine is the last tooth to be shed.

Caries did not develop in cats fed on any of the diets.

In the second generation of the cooked-meat cats the newborn deficient animal shows an irregular development of the contours of the skull cap and a narrowing of the malar and orbital arches, the latter becoming incomplete as deficiency progresses. Likewise the configuration of the mandible and

TABLE III. MILK EXPERIMENT NUMBER III

(In addition to the regular diet, each cat daily received approximately 15 mg. iron, 0.16 mg. copper, and 0.016 mg. manganese. Note the shortness of life of the metabolized vitamin D milk-fed males on the sole diet of milk)

NUMBER AND ORDER	AGE AT BEGINNING OF EXPERIMENT	LENGTH OF LIFE AFTER EXPERIMENT BEGAN	CALCIUM AND PHOSPHORUS RATIO	AVERAGE LENGTH OF LIFE
		Metabolized Vitamin D Milk		
Male				
*512 2C1	2 years 7 months	5 months	2.43	3 months
514 2C2	2 years 4 months	10 months	2.08	4 days
†594 2 Re L1	7 months 3 weeks	1 month 3 weeks	2.72	
620 1 Re L1	7 months	2 months	2.27	
651 Re	9 months	2 months 2 weeks	2.74	
654 Re	5½ months	1 month 10 days	2.11	
661 Re	6 months	1 month 19 days	2.36	
674 Re	Lost			
Female				
519 1C	3 years 6 months	3 months 1 week	3.00	4 months
586 1 Re L2	8 months	2 months 1 week	3.20	1 day
609 Re	Adult	8 months	2.69	
682 Re	7½ months	3 months 1 week	2.22	
		Pasteurized Milk		
Male				
511 2C1	2 years 6 months	3 months 12 days	2.19	4 months
515 2C2	2 years 5 months	10 months 1 week	2.23	11 days
531 2 Re L1	2 years 8 months	5 months	2.09	
589 Re	1 year 6 months	5 months	2.31	
592 2 Re L1	6½ months	2 weeks	2.13	
618 1 Re L1	7½ months	2 months 1 week	3.00	
658 Re	6½ months	2 months 14 days	4.70	
660 Re	6 months	1 month 11 days	2.31	
Female				
518 1C	3 years 8 months	4 months	2.33	3 months
521 R	2 years 4 months	2 months 3 days	2.38	27 days
566 Re	2 years 4 months	10 months 1 week	2.36	
584 1 Re L2	8 months	2 months	2.18	
655 Re	5 months	1 month 7 days	2.11	

*2C1 = Second generation, cooked-meat-fed animal, first litter.
†2 Re L1 = Second generation, regenerating animal, first litter.

maxilla is altered. As the kitten grows and develops, these abnormalities become more prominent. The eruption of the teeth is frequently followed by bleeding gums and prostration. Even the deciduous teeth are irregular in size and shape and are apt to be delayed in shedding. Supernumerary and missing teeth are common. Teeth do not erupt at a regular time as they do in the raw-meat-fed cat. Most of the cooked-meat-fed cats show a longer and narrower face with a retraction in the middle third. There is frequently a recessive mandible or a protruding mandible.

The permanent teeth are, in general, more irregular in size and alignment than the deciduous teeth. Gingivitis persists, gums become spongy, and abscesses gradually develop. It is through this process of secondary infection that most cats on a cooked-meat diet lose their teeth (Figs. 20*A*, 20*B*, and 20*C*). One of the most interesting factors is that the teeth in the second generation cooked-meat and regenerating cats frequently showed root resorption (Figs. 18*A*, 18*B*, and 18*C*). Becks[6] has suggested thyroid deficiency as a cause of root resorption. Although thyroid deficiency is sometimes demonstrated in

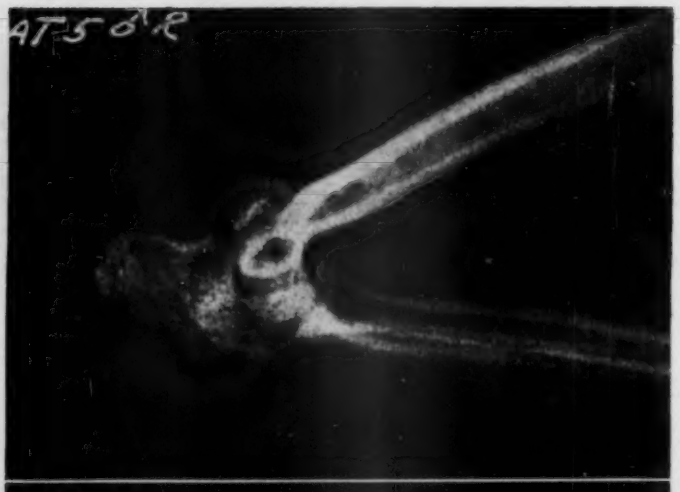


Fig. 16*A*.



Fig. 16*B*.



Fig. 16*C*.

Fig. 16*A*.—Elbow of male rat 5, fed on raw milk. Note advanced maturity, greater diameter and length of olecranon process of the ulna and finer internal trabeculation, as compared with Figs. 16*B* and 16*C*. All rats fed on milk from the same milkings.

Fig. 16*B*.—Elbow of male rat 9, fed on pasteurized milk. Note smaller olecranon process and delayed maturity when compared with rat 5.

Fig. 16*C*.—Elbow of male rat 44, fed on milk boiled five minutes. Note much coarser internal trabeculation of the olecranon process than in either Fig. 16*A* or Fig. 16*B*. Smaller in diameter, shorter in length.



Fig. 17*A*.—Lateral photograph of the skull of cat 539, primarily a raw-meat-fed animal that had had a short period of time (less than six months) on a cooked-meat diet. Note healed portion of alveolar processes around teeth previously damaged by the cooked-meat diet. Well-developed condyle places lower border parallel with upper jaw.



Fig. 17*B*.—Base of skull of cat 539, showing well-developed malar and orbital arches. Fair trabeculation.



Fig. 17*C*.—Lateral x-ray of half jaw of cat 539, showing a normal jaw structure, good distribution of trabeculae, well-developed condyle, and well-developed pterygoid process of the mandible. Alveolar crest of normal height; even distribution of teeth.

these animals, it is by no means constant. However, they all received a diet of denatured proteins which may play an important part in root resorption as well as chronic infections and disturbances in mineral metabolism. Loss of teeth in the deficient animal has come about as a result of paradentosis. Loss of vertical trabeculation and erosion of the alveolar processes occur in these animals.

It is interesting to note that root resorption occurred more commonly among the cats fed a diet of heat-processed milk than among those receiving cooked meat.

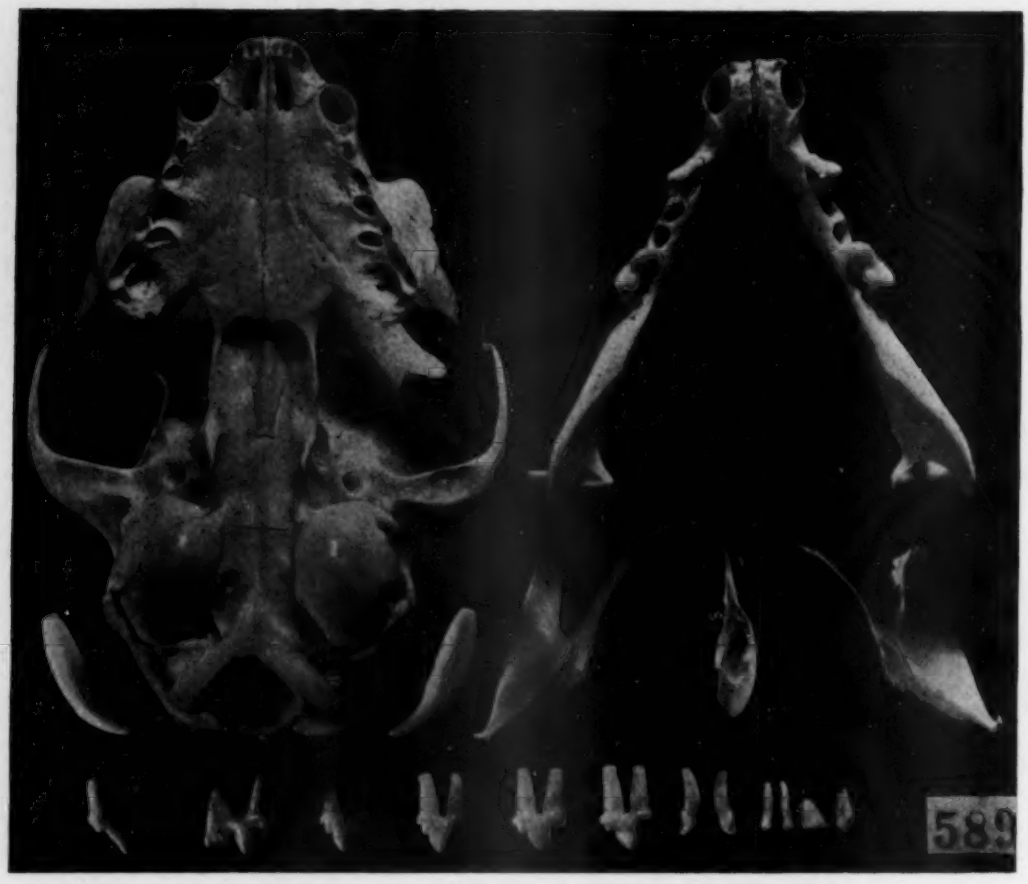


Fig. 184.—Regenerating male cat 589 on raw meat until 1 year, 2 months of age, following which he was on pasteurized milk experiment for four months. Note missing teeth, chalky appearance of bone, squaring off of bases of teeth and marked root resorption. Osteoporosis. Lack of completion of orbital arches. Malar bones have become separated at suture lines.

The degenerative changes in the skull and mouth grew more pronounced in the third generation cooked-meat cats (Figs. 10 and 11). The bones were very fine, with scarcely enough structure to hold the skull together. The teeth were smaller and much more irregular. When the permanent teeth erupted, the third generation cats were frequently prostrated.

In the regenerating cats, skull development is still deficient in the second generation, with a universal malalignment of teeth. The third generation



Fig. 18*B*.—X-ray of base of skull of regenerating male cat 589. Note lack of orbital and zygomatic arches and marked osteoporosis.

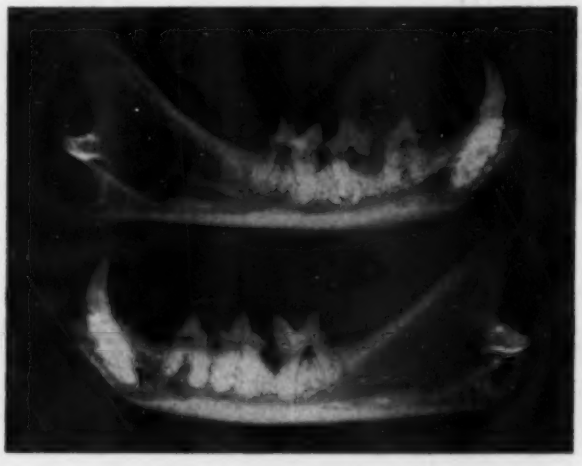


Fig. 18*C*.—X-ray of mandibles of regenerating male cat 589. Note marked osteoporosis and atrophy of the alveolar processes with small condyle head and poorly developed pterygoid process.



Fig. 19*A*.—First generation female cat 518, nursed one month, on a cooked-meat diet for three years, three months. She was then placed on the pasteurized milk experiment and died three months later. Note flattening of the entire skull with poor development of the condylar fossae. Distance from the zygomatic arch to the lower border of the mandible is lesser in the posterior jaw than the anterior in the region of the premolar teeth. Well-developed paradentosis with vertical atrophy. Poor distribution of trabeculae. Erosion of the condyle. Root resorption present in both upper and lower teeth.



Fig. 19*B*.—First generation female cat 518, 3 years, 7 months, as in Fig. 19*A*. Note partial resorption of orbital arches and poor structure of zygomatic arch. Evidence of osteoporosis. Poor distribution of trabeculae.



Fig. 19*C*.—First generation female cat 518, as in Fig. 19*A*. X-rays of mandible show root resorption, horizontal atrophy, poor internal structure, and atrophy of pterygoid process and condyles.



Fig. 20*A*.—Second generation male cat 513, second litter, cooked meat. Note apparently good skull except for incomplete orbital arch, resorption of the condyle, and the pulling up of the posterior portion of the mandible in comparison with the anterior portion.



Fig. 20*B*.—X-ray of basal view of skull of second generation male cat 513, as in Fig. 20*A*. Note lack of development of orbital arch and osteoporosis.

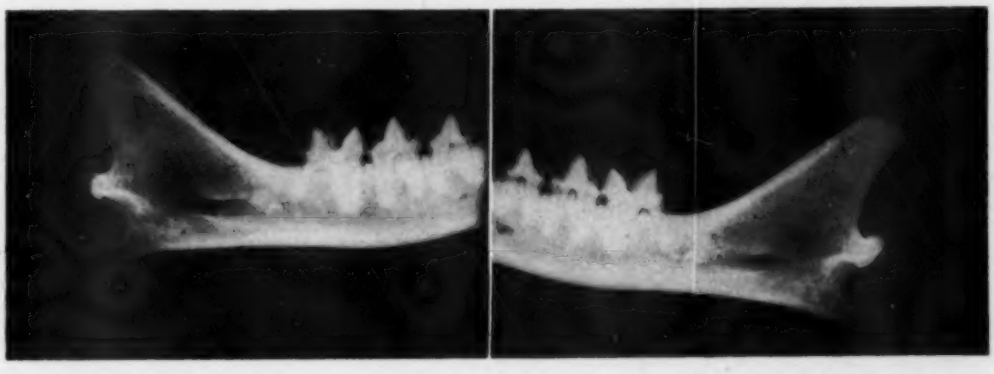


Fig. 20*C*.—X-rays of mandible of male cat 513, as in Fig. 20*A*. Note extreme osteoporosis, with almost complete loss of trabeculae, and failure in development of the heads of the condyles.

regenerating cats show improvement, and in the fourth generation, an occasional cat will show normal skull and dental development.

After we performed these experiments, the pens in which all of these animals were housed lay fallow for several months. Weeds sprang up in each pen. The fact that the weeds grew so luxuriantly in the pen which housed the raw-meat and raw-milk-fed animals, as compared with those which grew in the other pens, led us to perform a further experiment. We planted two kinds of beans in each pen. The results of the soil-fertilizing effect of the milk-fed female cats is shown in Fig. 21, where *A*, *B*, *C*, and *D* indicate the relative growths of vegetation in pens formerly occupied by cats on the various milk diets.

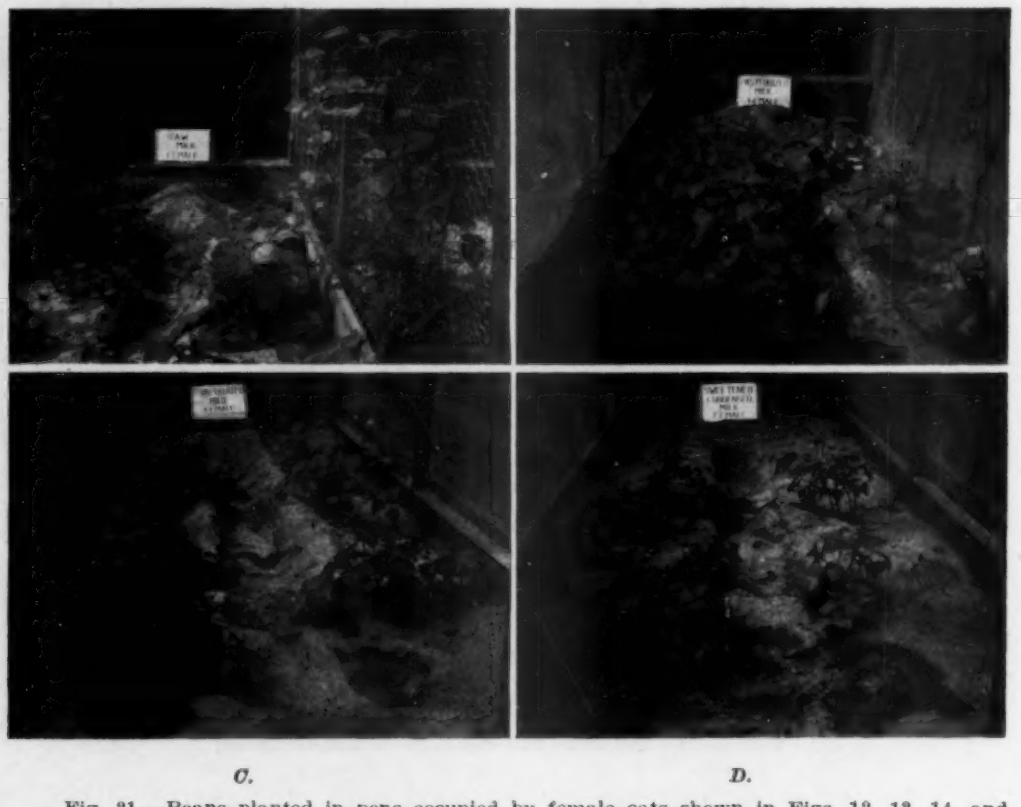


Fig. 21.—Beans planted in pens occupied by female cats shown in Figs. 12, 13, 14, and 15. *A*, Raw milk fed. *B*, Pasteurized milk fed. *C*, Evaporated milk fed. *D*, Sweetened condensed milk fed.

What vital elements were destroyed in the heat processing of the foods fed the cats? The precise factors are not known. Ordinary cooking precipitates proteins,[7,8] rendering them less easily digested.[9] Probably certain albuminoids and globulins are physiologically destroyed.[7, 10] All tissue enzymes are heat labile and would be materially reduced or destroyed. Vitamin C and some members of the B complex are injured by the process of cooking. Wulzen and Van Wagtendonk[11] have described a thermolabile substance in milk that may be one of the factors. Minerals are rendered less soluble by

altering their physicochemical state.[12] It is possible that the alteration of the physicochemical state of the foods may be all that is necessary to render them imperfect foods for the maintenance of health. *It is our impression that the denaturing of proteins by heat is one factor responsible.*

The principles of growth and development are easily altered by heat and oxidation, which kill living cells at every stage of the life process, from the soil through the plant, and through the animal. Change is not only shown in the immediate generation, *but as a germ plasm injury which manifests itself in subsequent generations of plants and animals.*

I wish to thank those who have contributed to this work: Dr. Allison James of Los Angeles, who first brought the dental aspects of the experiments to my attention; Dr. J. E. Ziegler, of Los Angeles, who has assisted with the x-rays of the skulls; Dr. D. G. Simonsen, also of Los Angeles, who supervised the care of the animals and the compiling of records; and the Lee Foundation for Nutritional Research, of Milwaukee, which has contributed other valuable assistance in the preparation of the article.

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FRAGMENTATION AND SCARRING OF THE TARSAL AND METATARSAL BONES: AN INDEX OF DENTAL DEFORMITY

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IN MY previous paper,* I described our experience with feeding experimental animals diets which produced deficiencies in skeletal and organic development. I demonstrated that these animals returned deficient material to the soil, which in turn produced deficient plants. In these experiments, we were working with only a part of the cycle which governs growth and development.



Fig. 1.—No. 4272(30). Skull of female, aged 6 weeks. Note mandibular protrusion and small middle third of face.

The entire cycle consists of soil to plant, to animal, and to man; then back to the soil. Primitive man of optimum development maintained this cycle. Anthropologists have pointed out that modern man has broken the chain, in-

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terfering with the regular homogeneous development of his kind. Price[1] has made an extensive study of "modern primitives" who have quickly shown degenerative changes on the food of civilized man. Wrench[2] has studied the Hunzas with the same general conclusion. McCarrison[3] has studied the diets of the men of India by feeding the identical foods to the rat, the pigeon, and the monkey. His results corresponded closely with those which we experienced with our own cats and rats.



Fig. 2.—No. 4272(46). Skull of female, aged 2 years, 5 months. Note increasing prominence of mandibular protrusion with failure of base of skull and middle third of face to move forward.

We who are practicing medicine and you who are practicing dentistry are confronted daily with the problems brought about by modern methods of agriculture, food processing and preparation, and by the deficient health of parents. If we are to bring intelligent help to our patients, we must employ all of the available information on growth and development that is yielded by a study of the sciences dealing with the health of man.

In this paper, it is my purpose to show you that we have been able to learn a great deal about the effect of malnutrition on the human being from x-ray studies. Just as we found that the shape of the skull of the newborn kitten was an index of its future health, so have we found that the skull of the newborn babe is likewise an index to the future health of the child.



Fig. 3.—No. 4272(58). Skull of female, 5 years, 1 month of age. Mandible still prominent. Sphenoid sinuses beginning to pneumatize. Middle third of face still small. Frontal sinuses not yet developed. Dental alignment fair.

Todd[4] called the skeleton the historian of the body. He pointed out that the scoring on the head of the radius and the long bones of the body represented metabolic upsets. In our own patients, we have found that the allergic child shows marked bone damage in the bones under stress at the time they are experiencing their most rapid growth. Fragmentation of the bones of the lower extremities occurs most commonly in the child in the period of life from 3 months up to approximately 6 years of age (Figs. 1 through 9).



Fig. 4.—No. 4272(32). Foot of female, aged 3 months. Marked angulation and concavity of the distal end of metatarsals 2, 3, 4, and 5. Slight condensation of the internal trabecular structure of the cuboid.



Fig. 5.—No. 4272 (47). Foot of female, aged 2 years, 5 months. Slight fragmentation of cuneiform 1 and 3. Mild roughness of navicular. Some roughness and fragmentation of bases of metatarsals of 2, 3, 4, and 5. No epiphyseal head of metatarsal 5. Metatarsal 4 shows epiphyseal head just appearing. Three is fragmented. Two is poorly developed. Proximal epiphysis of metatarsal 1 is rough.

The past few years, orthodontists have sought to correlate the structures of the mouth with some other structures of the body and to determine skeletal maturation. They have commonly elected to use the hand for this correlation. I propose to show you studies which will demonstrate that an ideal structure for the orthodontist to study in this connection is the foot.



Fig. 6.—No. 4272(59). Foot of female, aged 5 years, 1 month. Largely healed. Slight condensation at base of metatarsals. A little roughness of cuneiform 1.

We routinely take x-rays of the lateral and anteroposterior views of the skull as well as x-rays of the hand and foot of each patient. In the child, we make a particular study of the foot. The bones of the foot in the infant and growing child to 6 years of age are under the stress of beginning to walk (Figs. 10 through 29). This corresponds with the time of the erupting and beginning of shedding of deciduous teeth. At this time the dental arches are



Fig. 7.—No. 4272(31). Hand of female, aged 3 months. Well-developed hand; compares favorably with Todd Standard 1. Slight roughness of capitate.



Fig. 8.—No. 4272(45). Hand of female, aged 2 years, 5 months. Slight condensation of trabeculae in capitate and hamate. Near Todd Standard 8, except lunate not yet seen.

expanding to make room for the permanent dentition, while the buds of all the permanent teeth, with the exception of the third molars, are under way. At the same time the heads of the metatarsal bones are developing, epiphyses are taking shape, and the general contours of the cuboid bone are being developed. The navicular bone is forming, and the three cuneiform bones are being laid down. It is also the period in which the bases of the metatarsals are smoothing out and are completing their articulation with the cuboid and cuneiform bones and with each other. They are reaching an oval shape, while the general outline of the tarsal bones is almost completed.



Fig. 9.—No. 4272(57). Hand of female, aged 5 years, 1 month. Development slightly ahead of age. Slight internal condensation of capitate and hamate. Compares with Todd Standard 14: 5 years, 9 months.

Though the foot is the easiest of all the structures of the lower extremities to study, the ankle, the knee, and the hip may show disturbances in growth equally well. Although a child may show fragmentation such as is shown in the accompanying illustrations, he may present no external evidence of this problem. However, he may have flat feet or may limp slightly for a time.

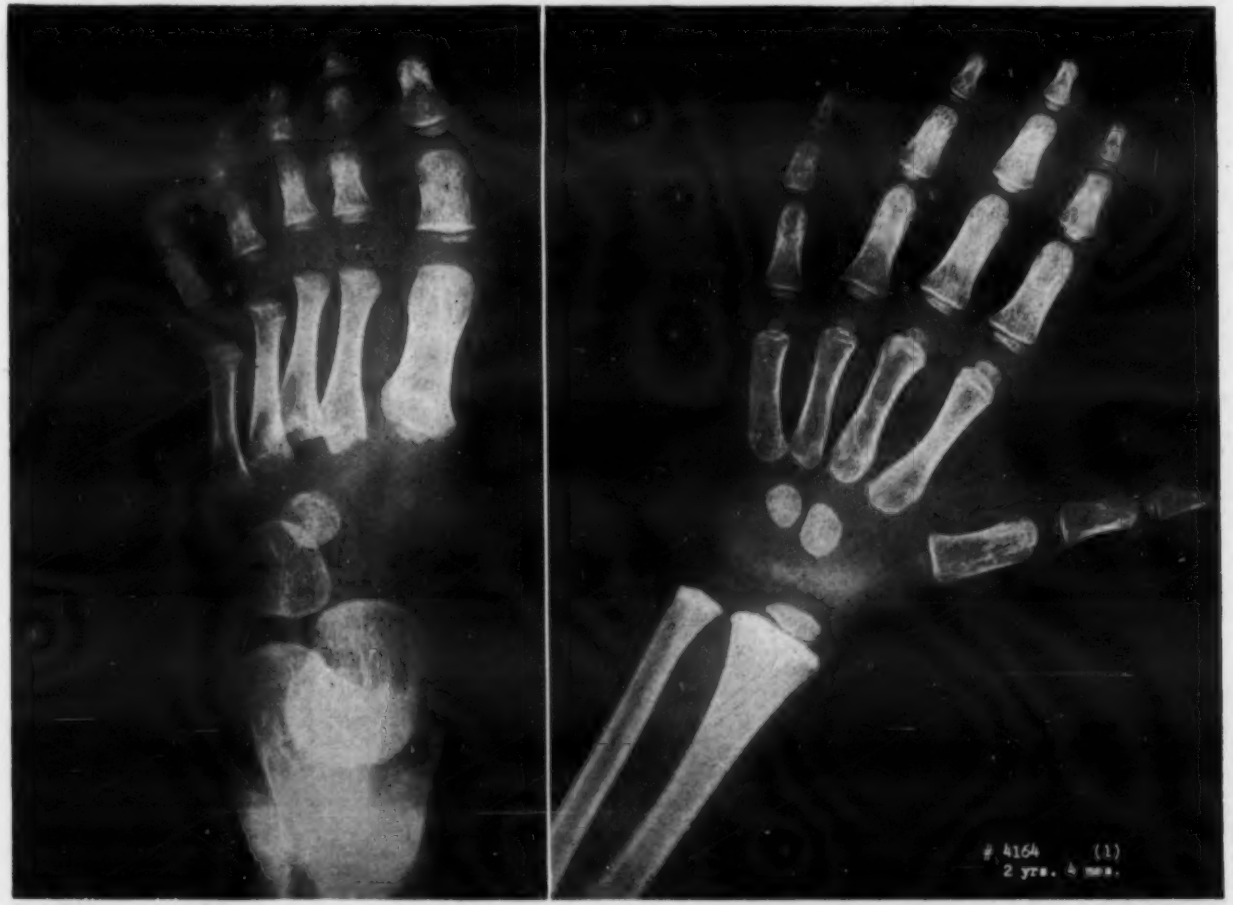


Fig. 10. Fig. 11.

Fig. 10.—No. 4164(2). Foot of male, aged 2 years, 4 months. Marked fragmentation of the bases of the metatarsals of 2, 3, 4, and 5, and proximal epiphysis of metatarsal of phalanx 1. Navicular delayed. Cuneiform 1 sligh ly fragmented. Flattening and irregularity of the heads, and fa.lure to develop of epiphyses of metatarsals 2, 3, 4, and 5.

Fig. 11.—No. 4164(1). Hand of male, 2 years, 4 months. Coarse trabeculation in all carpal and metacarpal phalangeal bones. Between Todd Standard 7 and Standard 8: 2 years, 3 months, and 2 years, 9 months.

Evidence of such a period of damage may occur in the "normally" healthy child when he experiences an intercurrent infection such as measles or any of the usual illnesses of childhood. This child may heal the damage with no one the wiser unless x-ray pictures are made at the time.

The first evidence of fragmentation of the bones of the foot of the allergic child may be seen as early as the third month. It reaches a maximum between the third and fourth year. This fragmentation is early noticeable in the epiphyses and heads of the metatarsals, which may be delayed in appearance.



Fig. 12.—No. 4164(3). Foot of male, aged 2 years, 10 months. Note fragmentation of bases of metatarsals. Increased fragmentation of proximal heads of metatarsal 1. Evidence of disturbance in mesial distal head of metatarsal 1; beginning fragmentation at head of metatarsal 1. Failure of heads to develop on metatarsals 3, 4, and 5. Cuneiform 1 fragmented. Cuneiform 2 fragmented. Cuneiform 3 rough and fragmented. Some internal condensation.

Fragmentation of the bases of the metatarsals is often present, and the navicular, cuboid, and three cuneiform bones may be laid down with many fragmented centers of ossification. These tend to heal so that, by 6 years of age, only the scars resulting from fragmentation are present. These scars consist of a thickening or internal condensation of the trabeculae in the tarsals and

the bases of the metatarsal bones. Later on, however, at about 10 to 12 years, as the epiphysis at the base of the fifth metatarsal appears, a new evidence of fragmentation in the foot may be seen (Fig. 38).

During this entire period, with the exception of certain scarring in the carpal bones that is represented by thickening or condensation of the internal trabeculation, no appreciable evidence of bone damage is found in the hand. In spite of profound disturbances which may be seen in the tarsal and metatarsal bones, bone age delay in the hand does not necessarily follow.



Fig. 13.—No. 4164(5). Hand of male, aged 2 years, 10 months. Essentially normal hand.

In the three cases which I am presenting, the carpal development of the patients is very close to the standards of Todd. When one finds evidence of fragmentation in the tarsal and metatarsal bones, it is only necessary to look at the face and into the mouth to see that other growth damage is also taking place. In some, it will be disturbance in growth of the middle third of the face alone. In others, it may be primarily a disturbance in expansion of the mandible. Some may have a combination of the two. Likewise, when evidences of severe metatarsal fragmentation are seen, one usually finds additional evidence of deficiency in the form of poor osseous structure and coarse trabeculation of the jawbone. This same disturbance in the trabecular pattern is apparent in the osseous structure of the entire body as well as in the foot and the hand. If the orthodontist wishes to obtain x-ray evidence from other





Fig. 14.

Fig. 15.

Fig. 14.—No. 4164(7). Foot of male, aged 3 years, 4 months. Note extensive fragmentation of metatarsal bases continuing, with slight amount of healing of metatarsal 5. Continued fragmentation of 2, 3, and 4. Appearance of metatarsal heads still delayed. Proximal epiphysis of metatarsal 1 still fragmented. Metatarsal head of 1 shows increased condensation. Internal condensation noted in cuboid and third cuneiform. Fragmentation of cuneiforms 1 and 2, and navicular, still prominent.

Fig. 15.—No. 4164(8). Hand of male, 3 years, 4 months of age. Capitate and hamate still show internal condensation and coarse internal trabeculation of the bones. Otherwise essentially normal.

Fig. 15.—No. 4164(8). Hand of male, 3 years, 4 months of age. Capitate and hamate still show internal condensation and coarse internal trabeculation of the bones. Otherwise essentially normal.





Fig. 16. Fig. 17.

Fig. 16.—No. 4164(11). Foot of male, aged 3 years, 10 months. Metatarsal 5 shows continued healing of the base. Healing just begun in metatarsal 4. Base of metatarsals 2 and 3 still clearly fragmented. Metatarsal head of 2 beg.nning to develop. Metatarsal 3 bifid; 4 and 5 still delayed. Frank osteochondrosis noted at the head of metatarsal 1, and proximal epiphysis still fragmented. Navicular and cuneiforms 1 and 2 still fragmented. Cuneiform 3 largely healed.

Fig. 17.—No. 4164(9). Hand of male, aged 3 years, 10 months. Essentially normal.



Fig. 18.



Fig. 19.

Fig. 18.—No. 4164(12). Foot of male, 4 years, 4 months of age. Metatarsal bases showing signs of healing. Greatest fragmentation noted in 3. Proximal epiphysis of 1 largely healed. Osteochondritis in the head of 1 continuing active. Epiphysis of 2, developing fairly normally. Epiphysis of 3 still bifid. Epiphysis of 4 appearing fragmented; epiphysis of 5 not yet present. Navicular and first cuneiform still fragmented; second cuneiform and third cuboid showing signs of healing.

Fig. 19.—No. 4164(14). Hand of male, aged 4 years, 4 months. Progress essentially normal.





Fig. 20. Fig. 21.

Fig. 20.—No. 4164(17). Foot of male, aged 4 years, 10 months. Bases of 4 and 5 essentially healed. Note internal condensation in bases. Two and 3 still healing with internal condensation evident. Proximal epiphysis of 1 largely healed. Distal portion of 1 shows osteochondritis still present. Epiphysis of 2 fairly well developed; 3 still bifid. Four came in as hemiepiphysis. Five not yet present. Second cuneiform almost healed. Navicular and first cuneiform healing. Internal condensation in all distal bones.

Fig. 21.—No. 4164(15). Hand of male, 4 years, 10 months of age. Condensation of capitate and hamate. Todd Standard 12: 4 years, 9 months.



Fig. 22. Fig. 23.

Fig. 22.—No. 4164(20). Foot of male, 5 years, 3 months of age. Bases of all metatarsals healing. Showed considerable condensation. Osteochondritis of first metatarsal healing. Epiphysis of second essentially normal. Epiphysis of third slightly bifid; head of 4 still a hemiepiphysis; 5 just beginning. Navicular and first cuneiform still rough. Second and third cuneiform and cuboid show internal condensation.

Fig. 23.—No. 4164(18). Hand of male, aged 5 years, 3 months. Essentially normal. Todd Standard, 5 years, 3 months.



Fig. 24.



Fig. 25.

Fig. 24.—No. 4164(23). Foot of male, aged 6 years. Navicular and first cuneiform still slightly rough. Marked internal condensation in all anterior tarsal bones. Marked internal condensation in bases of metatarsals. Head of metatarsal 1 shows osteochondritis nearly healed. Epiphysis on second metatarsal well developed. Third still partially developed; fourth less well developed than third, and fifth is fragmented.

Fig. 25.—No. 4164(21). Hand of male, aged 6 years. Internal condensation of capitate, hamate and triquetrum in evidence. Essentially normal development.



Fig. 26. Fig. 27.

Fig. 26.—No. 4164(26). Foot of male, aged 6 years, 11 months. Bases of metatarsals and tarsals essentially the same as in Fig. 25. Head of 1 shows improvement in ossification. Two and 3 fairly well developed. Four is improving and 5 is beginning to develop.

Fig. 27.—No. 4164(24). Hand of male, aged 6 years, 11 months. Essentially normal.



Fig. 28. Fig. 29.

Fig. 28.—No. 4164(29). Foot of male, aged 7 years, 8 months, now essentially normal except for internal condensation in the cuneiform, cuboid, and navicular bones, and the bases of the metatarsals. Slight evidence of healed osteochondritis in the first metatarsal head and flattening of the fifth metatarsal epiphysis.

Fig. 29.—No. 4164(27). Hand of male, aged 7 years, 8 months. Compares favorably with Todd Standard for 7 years, 9 months. With the exception of internal condensation shown in the capitate, hamate, triquetrum, and lunate, the hand is essentially normal.



Fig. 30.



Fig. 31.

Fig. 30.—No. 4209(62). Foot of female, aged 4 years, 11 months. Notice internal condensation in base of metatarsals, third cuneiform, navicular, and cuboid. Third cuneiform is apparently not quite healed.

Fig. 31.—No. 4209(60). Hand of female, aged 4 years, 11 months. Interior of capitate, hamate, and triquetrum shows condensation of trabeculation suggesting evidence of old injury. Bone study suggests Todd Standard 14: 5 years, 9 months.





Fig. 32. Fig. 33.

Fig. 32.—No. 4209(65). Foot of female, aged 5 years, 9 months. Same scars noted as in Fig. 30.
Fig. 33.—No. 4209(63). Hand of female, aged 5 years, 9 months. Same bones show condensation as in Fig. 31. Coarse trabeculation. Todd Standard 16: 6 years, 9 months.



Fig. 34.



Fig. 35.

Fig. 34.—No. 4209(68). Foot of female, aged 6 years, 10 months. Same as Fig. 32, except for growth.
Fig. 35.—No. 4209(66). Hand of female, aged 6 years, 10 months. Same scars noted as in Fig. 33. Todd Standard 18; 7 years, 9 months.





Fig. 36. Fig. 37.

Fig. 36.—No. 4209(71). Foot of female, aged 7 years, 8 months. Further increase in size. Pattern coarse.
Fig. 37.—No. 4209(69). Hand of female, aged 7 years, 8 months. Same. Very coarse trabeculation. Todd Standard 20: 8 years, 9 months.



Fig. 38.



Fig. 39.

Fig. 38.—No. 4209(74). Foot of female, aged 10 years, 2 months. Evidence of fragmentation in epiphysis of the tuberosity of the fifth metatarsal. All other scars carried on.

Fig. 39.—No. 4209(72). Hand of female, aged 10 years, 2 months. Evidence of internal condensation in metacarpal bases and all carpal bones. Todd Standard 25: 11 years, 3 months.



Fig. 40.



Fig. 41.

Fig. 40.—No. 4209(61). Skull of female, aged 4 years, 11 months. **Failure in forward movement of middle third of the face and base of skull, giving an apparent mandibular protrusion.**

Fig. 41.—No. 4209(64). Skull of female, aged 5 years, 9 months. Middle third and base of skull showing forward movement.



Fig. 42.



Fig. 43.

Fig. 42.—No. 4209(67). Skull of female, aged 6 years, 10 months. Further progress.

Fig. 43.—No. 4209(70). Skull of female, aged 7 years, 8 months. Malar prominence moving forward. Glabellar prominence beginning to develop.

portions of the body to correlate with his studies of the jaw, he should examine a bone or group of bones that is under stress at the time the dental structures are developing.

The scars that are laid down in these bones during periods of fragmentation are apparently carried on into adult life. We have been able to follow



Fig. 44.—No. 4209(73). Skull of female, aged 10 years, 2 months. Notice changing angle of upper teeth. Good alignment of teeth. Good forward projection of face and base of brain case. Series shows increase in growth of middle portion of face in respect to mandible.

evidences of this scarring as long as seven years after the process has largely healed (Figs. 30 through 46). In this one instance, we are showing you the process five years after the healing has taken place. We have not been studying the patients long enough to carry any single individual from infancy to adult life, so as to be able to say conclusively that a particular scar on a particular bone is evidence of a specific fragmentation that occurred in infancy.



Fig. 45.—No. 4209. Lateral views of patient at 4 years, 11 months of age, and 10 years, 2 months of age, showing changes in facial configuration and tissue stability.



Fig. 46.—No. 4209. Frontal view of patient at 4 years, 11 months of age, and 10 years, 2 months of age.



Fig. 47.—No. 4491. Skull of female aged 32 years, before improvement in dietary (left), and three years later (right), after giving birth to two children. Note changes in skull configuration and laying down of scar bone.

We have observed, however, that those of our adult patients who show dental and facial failures commonly acknowledged to be developmental in nature exhibit scars in their bones which are similar to those we have followed in our children.

When one states that these processes leave their scars even into adult life, he is apt to give the impression that he feels the individual is foredoomed to developmental failures that will follow him throughout his life. It is my impression at this time that it is impossible to remove the scars themselves. It is impossible to change the developmental patterns of bones laid down. However, if the proper forces of growth and development are applied in sufficient amount, material change in trend of growth can be obtained.

In the accompanying x-rays and photographs (Figs. 30 through 46) I am showing you a child who, at the age of 5 years, had a disfiguring mandibular protrusion. No orthodontic treatment was administered to this child, but the results obtained can be seen in photographs of her at the age of 10 years. She had been placed upon a vital diet rich in growth and developmental factors. She was encouraged to use her facial structures, by chewing tough objects, and by making faces. At the age of 4 years and 10 months, she had a profound mandibular protrusion; at the age of 10 years and 2 months, this had largely been corrected by the development of the middle third of the face.

I have selected this as representative of a group of children in which I have seen improvement in facial development due solely to improvement in intake of growth-promoting factors.

It is possible for the adult to rebuild calcium in his bones by consuming adequate quantities of growth-promoting factors. Relative efficiency of all the organs of the body can also be increased noticeably.

It seems that the new bone laid down in this process is not the original bone that was present before the damage occurred, but is a scar bone, as is shown clearly by x-ray (Fig. 47).

Regarding the development of the orbital and maxillary arches, we find the same factors at work in the human being as we found experimentally in the cat. In our allergic children we have found a narrowing of the transverse diameter between the malar prominences, a narrowing of the transverse diameter between the lateral orbital prominences, failure in the forward movement of the malar prominence; all of these, or a combination of any may occur in an individual.

Muscle tone is largely controlled by factors similar to those that control growth and development. The density and strength of bone is determined by its calcium content and the muscle pulls that are exerted on it. These, in turn, are controlled by the factors of growth and maturity. It is not just the chewing on rawhide that develops the face of the Eskimo and it is not just the consumption of large quantities of raw fish, blubber, and entrails that produces his well-developed features, but it is the combination of them all, plus a firm inheritance that has not been interfered with in the ancestry of these individuals.

Every structure of the body, including the generative organs, is involved in the developmental process. However, from the standpoint of the dentist, if he is to correlate the findings in the jaw with other osseous structures, he will find a parallel in the x-ray of the foot, which is an organ under stress during the critical period of growth and development of the dental structures of the child.

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NUTRITION IN ACTION

MICHAEL J. WALSH, M.Sc., F.R.I.C., A.I.CH.E., SAN DIEGO, CALIF.

THOSE of us who participated in the First Annual Seminar for the Study and Practice of Dental Medicine were keenly aware of the unfortunate fact that the meals served to the seminarians were utterly inadequate from the nutritional standpoint. We felt that eating such foods as were served was inappropriate for practitioners who were trained in principles of nutrition and who expected to apply these principles in the furtherance of preventive dentistry.

For the Second Annual Seminar it was decided that the menus should be supervised to such an extent that the meals served would conform to the highest possible nutritional standards. Arrangements were made to have the writer plan the menus so that the entire dietary furnished types and amounts of foods which would conform to the highest nutritional standards.

With the magnificant cooperation of the Desert Inn management, this feature of "Nutrition in Action" proved to be a vital and important part of the Seminar. The meals were planned with the following objectives:

a. To provide the seminarians with the maximum possible quantities of essential nutrients contained in the minimum number of calories. The base for these computations was adjusted to the requirements of practicing dentists, who normally have very little physical activity but who work under conditions of very high pressure and tension.

b. To serve foods suitable to the environment, which, in Palm Springs, California, is semitropical.

c. To demonstrate visually by means of the evaluated menus given to each seminarian what actually constitutes a 12 ounce serving of roast beef, a 6 ounce serving of vegetables, etc., and further to show what a given meal, when eaten, yields in total calories, grams of protein, fat, and carbohydrates, milligrams of minerals, and units or milligrams of the various vitamins.

d. To provide practical demonstrations of the diet which should be prescribed and recommended for patients in dental practice, such as for caries prevention, orthodontics, etc.

e. To provide within the five-day period of the Seminar such conditioning mechanisms as the absence of sugar, ice cream, pies, etc., from the meals, and thereby to convince the seminarians that faulty diet habits built around the excessive use of refined sugar and refined grains can be rapidly and painlessly corrected by the substitution of foods more desirable from the nutritional standpoint.

Report on the diet status of the meals served at the Second Annual Seminar for the Study and Practice of Dental Medicine, by the Desert Inn, Palm Springs, California, Oct. 7 to Oct. 12, 1945.

The nutritional objective was to furnish daily, in the meals served, 2500 calories containing 225 grams of protein (predominantly of animal origin), 2 grams of calcium, and correspondingly optimum amounts of the other essential minerals and essential vitamins.

Evaluation of the menus established that the food served daily contained the following average amounts of nutrients:

Protein	220 gms.	Iron	27 mgs.
Fat	117 gms.	Iodine	230 mmgs.*
Carbohydrates	184 gms.	Vitamin A	15,500 I.U.
Calories	2,680	Thiamin	1,590 mmgs.*
Calcium	2,280 mgs.	Riboflavin	4,420 mmgs.*
Phosphorus	3,070 mgs.	Nicotinic Acid	56 mgs.
Ca:P Ratio	1:1.3	Ascorbic Acid	180 mgs.

Vitamin D 166 I.U.

Detailed evaluations of every meal were given on the menus; a complete set of these was furnished to each seminarian to serve not only as a valuable, practical lesson on "Nutrition in Action," but also to be retained as a happy souvenir of the occasion. Figs. 1 to 13, inclusive, illustrate the actual menus as furnished for the five days, and show the quantities of nutrients obtained if the meals were eaten as served.

Dinner

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR
For the Study and Practice of Dental Medicine

Louisiana Shrimp Cocktail Supreme
French Onion Soup, Crouton
Hearts of Celery Carrot Sticks
Rose Radishes Green Onions
Roast Prime Ribs of Beef Au Jus
Baked Idaho Potatoes
Cauliflower Polonaise New Green Peas
Health Whole Wheat and Pumpernickle
Mixed Green Salad, Seminar Dressing
Delicious Apples Bartlett Pears
Valencia Oranges Bananas
American Cheddar, Swiss or Camembert Cheese
Toasted Bents or Saltines
Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE DINNER

This dinner was designed to supply the following amounts of nutrients:

Protein	123 gms.	Iron	15 mgs.
Fat	54 gms.	Iodine	68 mmgs.
Carbohydrates	112 gms.	Vitamin A	6000 I.U.
Calories	1430	Thiamin	720 mmgs.
Calcium	1280 mgs.	Riboflavin	1840 mmgs.
Phosphorus	1470 mgs.	Nicotinic Acid	28 mgs.
Ca:P Ratio	1:1.1	Ascorbic Acid	65 mgs.

Vitamin D 20 I.U.

This Menu compiled for the Dental Seminar by,
Michael J. Walsh, M.Sc., F.R.I.C., A.I.Ch.E.

Sunday, October 7, 1945

Fig. 1.

*Mmgs. = micrograms.

Breakfast

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR
For the Study and Practice of Dental Medicine

Half Grapefruit Maraschino
Orange or Grapefruit Juice
Cantaloupe, Honeydew or Persian Melon
Certified Ham and Eggs, Any Style
Whole Grain Breads
Coffee Milk

NUTRITIONAL FACTS ABOUT THE BREAKFAST

This breakfast was designed to supply the following amounts of nutrients:

Protein	53 gms.	Phosphorus	810 mgs.
Fat	27 gms.	Ca:P Ratio	1:1.6
Carbohydrates	53 gms.	Iron	8 mgs.
Calories	670	Iodine	20 mmgs.
Calcium	490 mgs.	Vitamin A	4800 I.U.
		Thiamin	390 mmgs.
		Riboflavin	1500 mmgs.
		Nicotinic Acid	17 mgs.
		Ascorbic Acid	77 mgs.
		Vitamin D	50 I.U.

Monday, October 8, 1945

Fig. 2.

Luncheon

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR
For the Study and Practice of Dental Medicine

Tomato Juice with Plain Gelatin
Cold Salmon Plate with Mixed Green Salad, Garni
Whole Grain Breads
Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE LUNCHEON

This luncheon was designed to supply the following amounts of nutrients:

Protein	41 gms.	Phosphorus	730 mgs.
Fat	23 gms.	Ca:P Ratio	1:1.9
Carbohydrates	32 gms.	Iron	4 mgs.
Calories	500	Iodine	140 mmgs.
Calcium	380 mgs.	Vitamin A	2150 I.U.
		Thiamin	320 mmgs.
		Riboflavin	780 mmgs.
		Nicotinic Acid	14 mgs.
		Ascorbic Acid	24 mgs.
		Vitamin D	450 I.U.

Monday, October 8, 1945

Fig. 3.

Dinner

NUTRITION IN ACTION

Souvenir Menu
of the
SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Crab Cocktail Supreme
Cream of Celery Soup

Mixed Olives — Carrot Sticks
Rose Radishes — Green Onions

Charcoal Broiled French Lamb Chops
Parsley Potatoes
Green Peas and Carrots
Tomato and Asparagus Tip Salad, Seminar Dressing
Fresh Mission Figs
American Cheddar, Swiss or Camembert Cheese
Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE DINNER

This dinner was designed to supply the following amounts of nutrients:

Protein	105 gms.	Phosphorus	1750 mgs.
Fat	113 gms.	Ca:P Ratio	1:1.2
Carbohydrates	88 gms.	Iron	14 mgs.
Calcium	1430 mgs.	Iodine	78 mmgs.
Calories	1780	Vitamin A	11,500 I.U.

Thiamin	620 mmgs.
Riboflavin	1860 mmgs.
Nicotinic Acid	13 mgs.
Vitamin D	5 I.U.
Ascorbic Acid	42 mgs.

Monday, October 8, 1945

Fig. 4.

Breakfast

NUTRITION IN ACTION

Souvenir Menu
of the
SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Orange Juice
Cantaloupe, Honeydew or Persian Melon
Grilled Sirloin Steak
Hashed Brown Potatoes
Whole Grain Breads
Coffee Tea Milk

NUTRITIONAL FACTS ABOUT THE BREAKFAST

This breakfast was designed to supply the following amounts of nutrients:

Protein	82 gms.	Phosphorus	680 mgs.
Fat	25 gms.	Ca:P Ratio	1:1.5
Carbohydrates	66 gms.	Iron	6 mgs.
Calories	820	Iodine	36 mmgs.
Calcium	440 mgs.	Vitamin A	2740 I.U.

Thiamin	530 mmgs.
Riboflavin	1250 mmgs.
Nicotinic Acid	20 mgs.
Ascorbic Acid	81 mgs.
Vitamin D	5 I.U.

Tuesday, October 9, 1945

Fig. 5.

Luncheon

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR
For the Study and Practice of Dental Medicine

Clam Juice Cocktail
Cold Sea Food Plate with Louis Dressing
Mixed Green Salad, Seminar Dressing
Health Breads
Rye Rolls

Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE LUNCHEON

This luncheon was designed to supply the following amounts of nutrients:

Protein	40 gm.	Phosphorus	700 mg.
Fat	6 gm.	Ca:P Ratio	1:1.7
Carbohydrates	22 gm.	Iron	3 mg.
Calories	300	Iodine	290 mmg.
Calcium	400 mg.	Vitamin A	850 I.U.

Thiamin	220 mmg.
Riboflavin	420 mmg.
Nicotinic Acid	11 mg.
Ascorbic Acid	13 mg.
Vitamin D	—

Tuesday, October 9, 1945

Fig. 6.

Dinner

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR
For the Study and Practice of Dental Medicine

Eastern Oyster Cocktail Supreme
Green Split Pea Soup

Mixed Olives Carrot Sticks
Celery Hearts Green Onions

Half Roast Spring Chicken
Potatoes au Gratin
Carrots Vichy
Whole Grain Breads
Avocado and Grapefruit Salad, Seminar Dressing
Fresh Boysenberries in Gelatine
American Cheddar or Swiss Cheese
Toasted Bents or Saltines

Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE DINNER

This dinner was designed to supply the following amounts of nutrients:

Protein	102 gm.	Phosphorus	1690 mg.
Fat	59 gm.	Ca:P Ratio	1:1.1
Carbohydrates	68 gm.	Iron	13 mg.
Calories	1200	Iodine	68 mmg.
Calcium	1480 mg.	Vitamin A	4680 I.U.

Thiamin	890 mmg.
Riboflavin	1370 mmg.
Nicotinic Acid	32 mg.
Ascorbic Acid	54 mg.
Vitamin D	25 I.U.

Tuesday, October 9, 1945

Fig. 7.

Breakfast

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Half California Grapefruit
Fresh Mission Figs
Grilled Calves Liver and Bacon
Whole Grain Breads
Coffee Tea Milk

NUTRITIONAL FACTS ABOUT THE BREAKFAST

This breakfast was designed to supply the following amounts of nutrients:

Protein	39 gm.	Phosphorus	600 mg.
Fat	22 gm.	Ca:P Ratio	1:1.4
Carbohydrates	57 gm.	Iron	11 mg.
Calories	580	Iodine	22 mmg.
Calcium	430 mg.	Vitamin A	10,200 I.U.
	Thiamin	520 mmg.	
	Riboflavin	3060 mmg.	
	Nicotinic Acid	25 mg.	
	Ascorbic Acid	58 mg.	
	Vitamin D	50 I.U.	

Wednesday, October 10, 1945

Fig. 8.

Luncheon

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Orange Juice with Plain Gelatin
Cold Meat Plate with Potato Salad
Sliced Tomatoes, Seminar Dressing
Rye Rolls
Plain Custard
Fresh Fruit

Coffee Tea Milk Buttermilk

NUTRITIONAL FACTS ABOUT THE LUNCHEON

This luncheon was designed to supply the following amounts of nutrients:

Protein	36 gm.	Phosphorus	610 mg.
Fat	34 gm.	Ca:P Ratio	1:1.3
Carbohydrates	73 gm.	Iron	6 mg.
Calories	750	Iodine	30 mmg.
Calcium	460 mg.	Vitamin A	3200 I.U.
	Thiamin	430 mmg.	
	Riboflavin	1120 mmg.	
	Nicotinic Acid	9 mg.	
	Ascorbic Acid	80 mg.	
	Vitamin D	—	

Wednesday, October 10, 1945

Fig. 9.

Dinner

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Seafood Cocktail Supreme
Cream of Asparagus Soup

Mixed Olives — Green Onions
Rose Radishes — Carrot Sticks

Roast Leg of Lamb, Mint Jelly
Rissole Potatoes

Summer Squash — Green Beans

Whole Grain Breads
Mixed Green Salad with Tomatoes
Seminar Dressing
Youngberries in Gelatine
American Cheddar, Blue, Camembert, or Swiss Cheese
Toasted Bents or Saltines

Coffee — Tea — Milk — Buttermilk

NUTRITIONAL FACTS ABOUT THE DINNER

This dinner was designed to supply the following amounts of nutrients:

Protein	120 gm.	Phosphorus	1490 mg.
Fat	52 gm.	Ca:P Ratio	1:1.3
Carbohydrates	99 gm.	Iron	11 mg.
Calories	1340	Iodine	59 mmg.
Calcium	1180 mg.	Vitamin A	11,300 I.U.
	Thiamin	590 mmg.	
	Riboflavin	1700 mmg.	
	Nicotinic Acid	14 mg.	
	Ascorbic Acid	69 mg.	
	Vitamin D	20 I.U.	

Wednesday, October 10, 1945

Fig. 10.

Breakfast

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Orange and Grapefruit Juice
Assorted Melons
Premium Ham and Eggs
Whole Grain Breads

Coffee — Milk

NUTRITIONAL FACTS ABOUT THE BREAKFAST

This breakfast was designed to supply the following amounts of nutrients:

Protein	53 gm.	Phosphorus	820 mg.
Fat	28 gm.	Ca:P Ratio	1:1.6
Carbohydrates	60 gm.	Iron	8 mg.
Calories	700	Iodine	21 mmg.
Calcium	500 mg.	Vitamin A	4700 I.U.
	Thiamin	410 mmg.	
	Riboflavin	1560 mmg.	
	Nicotinic Acid	17 mg.	
	Ascorbic Acid	90 mg.	
	Vitamin D	50 I.U.	

Thursday, October 11, 1945

Fig. 11.

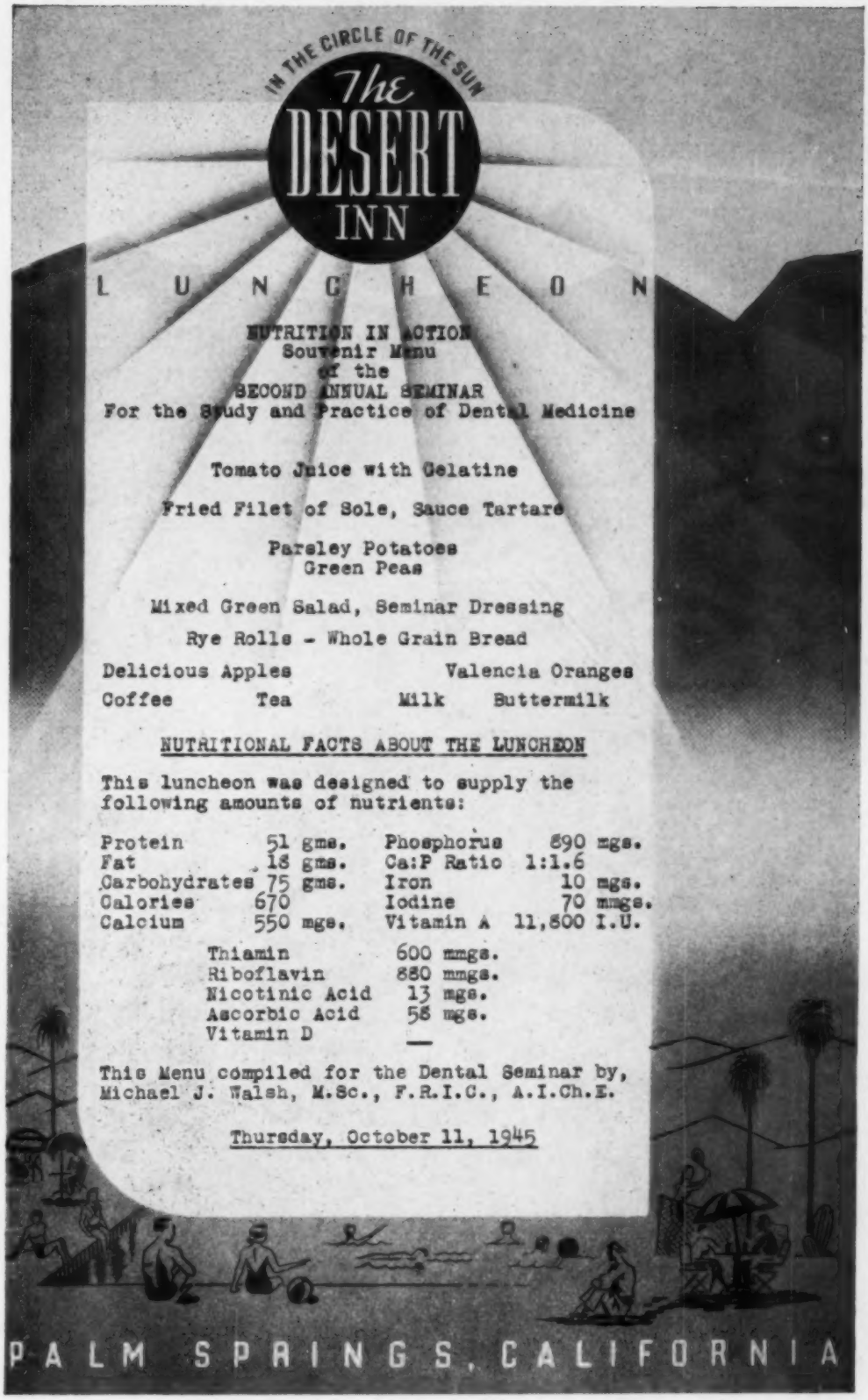


Fig. 12.—Reproduction of one of the thirteen souvenir menus.

Dinner

(Served at the Barbecue Pit)

NUTRITION IN ACTION

Souvenir Menu
of the

SECOND ANNUAL SEMINAR

For the Study and Practice of Dental Medicine

Louisiana Shrimp Cocktail Supreme
French Onion Soup, Crouton

Hearts of Celery	Carrot Sticks
Rose Radishes	Green Olives

Barbecued Rare New York Cut Steak
Baked Idaho Potatoes

Cauliflower Au Gratin	String Beans
Delicious Apples	Bartlett Pears
Valencia Oranges	Bananas

American Cheddar, Swiss or Camembert Cheese
Toasted Bents or Saltines

Coffee	Tea	Milk	Buttermilk

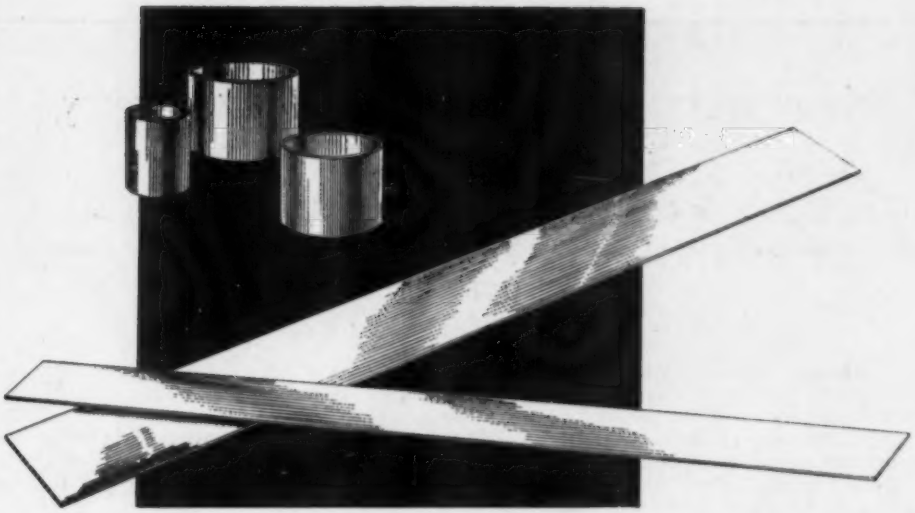
NUTRITIONAL FACTS ABOUT THE DINNER

This dinner was designed to supply the following amounts of nutrients:

Protein	157 gm.	Phosphorus	1700 mg.
Fat	77 gm.	Ca:P Ratio	1:1.1
Carbohydrates	110 gm.	Iron	13 mg.
Calories	1750	Iodine	74 mmg.
Calcium	1480 mg.	Vitamin A	6000 I.U.
	Thiamin	880 mmg.	
	Riboflavin	2300 mmg.	
	Nicotinic Acid	32 mg.	
	Ascorbic Acid	69 mg.	
	Vitamin D	9 I.U.	

Thursday, October 11, 1945

Fig. 13.



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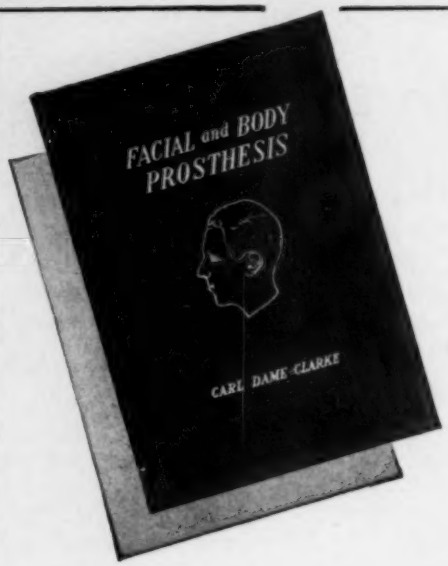
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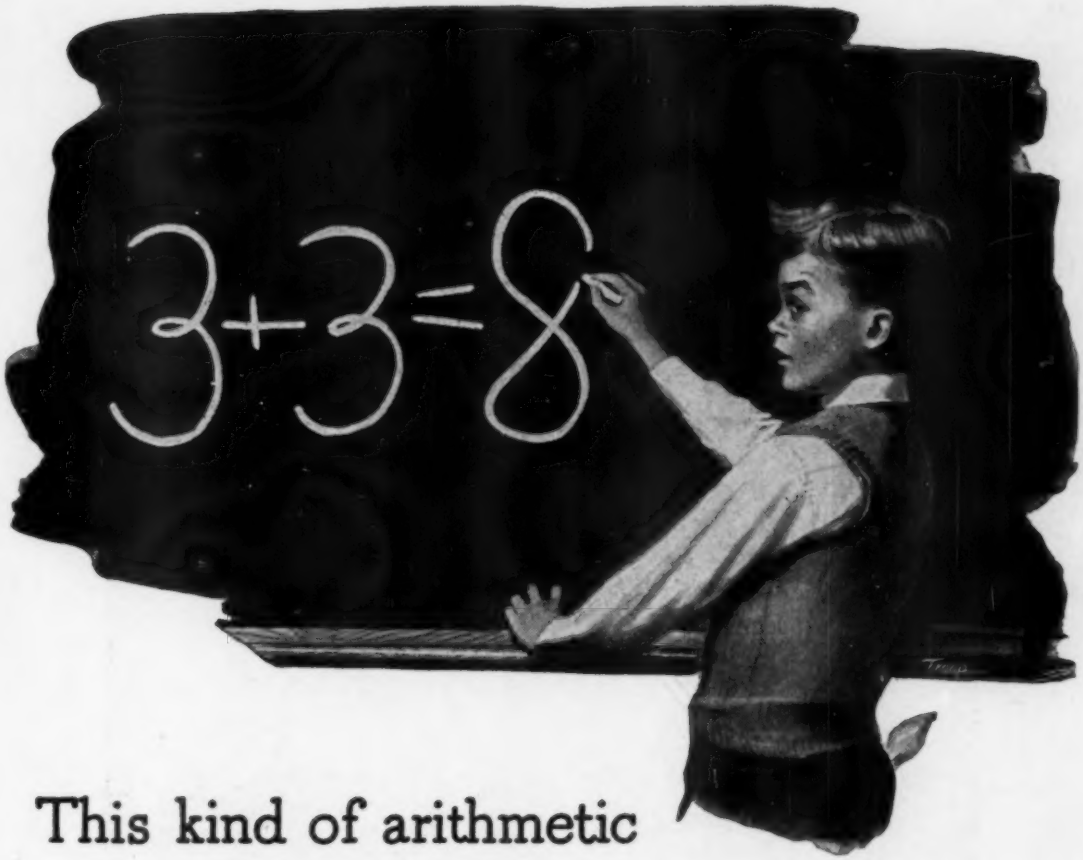
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Army Institute of Pathology Number

FOREWORD

THERE has been a cordial and profitable relationship between the Army Institute of Pathology and the dental profession for many years. The Institute, or Army Medical Museum as it was known in those days, was "formally adopted" by the American Dental Association in 1895 as its National Museum. For years the activity was largely restricted to that of a museum, and a considerable collection of historic and scientific material associated with dentistry was accumulated. A large room of the Museum was set aside for this collection and was one of the popular exhibits, not only to the profession but to the lay visitors. It contains a number of very valuable special collections, considerable comparative anatomy and much pathology. The enormous increase in work of the Institute incident to the war necessitated the storing of about half of the public exhibit, including much of the dental material, but it has all been very carefully safeguarded for the day when we have quarters appropriate for the expanded functions of the Institute. Then the dental collection will have a very prominent place and will be a great credit to the Institute, as well as to the dental profession.

The more recent emphasis on dental and oral pathology has opened up great possibilities in the relationship of the dental profession with the Institute. What has been accomplished and what is hoped to be accomplished have been outlined elsewhere. One of the most significant results is that dentistry, so far as the Institute is concerned, has become a specialty of medicine, just as is ophthalmology or chest surgery. It is felt that the Registry of Dental and Oral Pathology has been an important factor in elevating the professional standards of dentistry and that this possibility has by no means been entirely exploited. It is hoped that the practicing dentists may come to look to their Registry more and more for help in their clinical problems, that the dental schools will take more and more advantage of the material that has accumulated in the Registry, that those engaged in research will use the facilities of the Registry, and that all categories will come to look on their Registry as a depository for their cases and studies, for such centralization will enormously enhance their value.

J. E. Ash, Colonel, Medical Corps, U. S. Army, Director

THE ARMY INSTITUTE OF PATHOLOGY AND THE REGISTRY OF DENTAL AND ORAL PATHOLOGY

HENRY A. SWANSON, D.D.S.*

THE Army Institute of Pathology, formerly the Army Medical Museum, is recognized as a pivotal organization in the field of pathology.

The original function of the Army Medical Museum, when established in 1862 by order of Surgeon General William A. Hammond, was to collect anatomic specimens illustrating wounds and effects of projectiles. It was General Hammond's thought that the study of such material would "lead to a reduction in mortality." His vision has materialized through investigation and teaching by the staff members of this institution.

In 1937 there were 150,000 specimens and 45,000 photographs, including not only gross material but all types of pathologic material on file at the Institute. Since that time this has mounted to such an extent that the facilities have become overtaxed, and study of much of this material will, of necessity, await the time when increased personnel and facilities are provided.

The new title, "The Army Institute of Pathology," indicates more clearly the present functions of the Institute. Army regulations state that "the primary professional function of the Army Medical Museum is to furnish a central service for tissue pathology."

As constituted at present, this Institute has three principal functions: (1) it furnishes a consultation service for the diagnosis of pathologic specimens for the entire Army; (2) it conducts investigation and research on diseases of medicomilitary importance; and (3) it provides instruction in pathologic anatomy to Medical Department officers.

To accomplish these functions, the Institute is divided into four major units: (a) Laboratory of Pathology, (b) American Registry of Pathology, (c) Army Medical Illustration Service, and (d) Army Medical Museum.

That the Army Institute of Pathology has successfully carried out these functions during the past years is readily apparent, for through the very excellent work of the staff of pathologists and their associate workers it is now recognized not only by the Army medical officers, but among the civilian pathologists as well.

Army regulations further direct that the material collected be made available "for teaching purposes at Army Medical Department schools, other schools devoted to military education and for recognized medical, dental and veterinarian schools." The regulations also order the conduct of "investigations and research on the accessions" and the arrangement of material so "that it

*Chairman, Committee on National Museum and Dental Registry of the American Dental Association.

will be available for reference and study by other properly qualified investigators." It further authorizes the Institute "to act as custodian for the American Registry of Pathology of the National Research Council."

LABORATORY OF PATHOLOGY

The consultation service at the Institute for the diagnosis of pathologic tissue for the Army is very effective. This service has also been made available to civilian pathologists and clinicians, through the courtesy of the Surgeon General of the United States Army. During the period of world conflict it was utilized to the utmost by the Medical Department officers throughout the world. By air mail, autopsy and surgical material, together with complete histories, was sent in to the Institute; the pathologic interpretation and diagnosis was either air mailed or telegraphed to the submitting source. Speed was essential, and prompt handling of all material was imperative to save life or to prevent a serious medical catastrophe.

The staff stationed at the Institute consists of general pathologists and others trained in special fields, such as dental and veterinary medicine. Leading pathologists in civilian practice have been designated as consultants, and cases of unusual interest are referred to these men for what often proves to be a timely comment. Some of the consultants spend periods of a month or longer at the Institute engaged in special studies. With such close cooperation, the consulting service has become most valuable to the Army Medical Department and the civilian professional groups as well.

The study of the vast amount of material received at the Institute has disclosed some surprising and important facts. For instance, certain malignant tumors hitherto supposed to occur only in later life have been found to develop in the relatively young age group under special environmental conditions. Thus, among troops in certain areas, 200 carcinomas of the lip were recorded.

TEACHING

The teaching activities of the Institute have not been limited to the Army Medical Department but have been made available to medical, dental, and veterinary schools, to professional groups, and to individuals. Teaching material has been prepared in the form of study sets consisting of microscopic slides illustrating the pathology of various special fields, together with syllabuses giving a complete description of the material.

A number of valuable definitive studies have been published and are in preparation which have utilized not only the pathologic material, but also selections from the wealth of illustrative material at the Institute.

One of the most popular teaching aids which have been developed is the Atlases. To date there have been 7, each in a different field, and they are being used in professional schools, hospitals, and clinics throughout this country.

Lantern slides, 3¼ by 4 inches, in both color and black and white, and 2 by 2 inch slides, also in color and black and white, covering the special fields of pathology, have been prepared and may be obtained for teaching purposes.

No single medical, dental, or veterinary school has at its disposal the wealth and variety of pathologic material accumulated at the Institute. To those interested in pathology, the Institute is a valuable source of help in the preparation for special studies.

THE AMERICAN REGISTRY OF PATHOLOGY

The American Registry of Pathology, operating under the auspices of the National Research Council, is housed at the Army Institute of Pathology by authority of the Surgeon General of the United States Army. The purpose of the Registry of Pathology is to collect pathologic material, record and catalogue it, and to conduct comprehensive investigations. At present there are 14 registries, each representing a special field of pathology. They are sponsored by national scientific and professional associations, and this connection brings about close cooperation between these organizations and the Institute.

ARMY MEDICAL MUSEUM

The Army Medical Museum, which is open to the public, is, as the name implies, a veritable storehouse of interesting exhibits covering (a) material relating to the history of medicine, (b) trophies of the Army Medical Corps, and (c) anatomic specimens illustrating the process of disease. It is set up in such a way as to interest both professional and lay people and was visited by approximately 200,000 persons during the past year.

REGISTRY OF DENTAL AND ORAL PATHOLOGY

What part does dentistry have in this great institution? This question can be answered effectively, for dentistry has been given prominence equal to that of any other branch of medicine. Through the courtesy and help of the present Director, Colonel James E. Ash, the dental section has become most active and valuable. The American Dental Association inaugurated a Committee on Museum in 1931, and in 1933 authorized the Registry of Dental and Oral Pathology to be under direction of that committee.

The Mid-Monthly *Journal of the American Dental Association* for April, 1946, carried a report of the activities of the Museum Committee, which deals with the work of the dental section of the Army Institute of Pathology. The work of the Registry has progressed most satisfactorily.

Army dental installations, both in this country and abroad, forward to the Institute all surgical material with clinical records for diagnosis, and much of this material is included in the Registry. Through the cooperation of the various members of the Museum Committee, the dental pathologists associated with various dental schools, the designated consultants, and many supporters of the Registry, material is constantly being received, so that there are 5940 cases registered at present.

Many of these cases are contributed by dental pathologists and oral surgeons who realize the value of a centralized registry in making material available for study and investigation. All dental and oral cases sent to the Institute are evaluated and those meeting the criteria are accepted for the Registry and

given an accession number under the name of the contributor. Biopsy, diagnostic, and consulting service is available to all practitioners of dentistry, and most cases received for diagnosis are also included in the Registry. Contributors are urged, however, to submit cases through their local pathologic laboratory, if one is available.

The general practitioner of dentistry often has the first opportunity of observing lesions in the oral cavity and it is his responsibility to detect early conditions which might endanger the life of the patient. Whether he operates or refers the patient to a surgeon, he can be assured that the Institute will furnish consultation service if tissue is removed.

Practically all of the diagnostic work of the Registry of Dental and Oral Pathology is concerned with tumors of the mouth and adjoining areas. However, simple lesions of the teeth and supporting structures, in fact any dental pathologic material, are welcomed because the greater the accumulation the more valuable is the Registry for study.

No attempt will be made here to list all the types of lesions, but the statistical analysis of the cases in the Registry shows several large collections, including: 75 ameloblastomas, 410 radicular cysts, 750 mixed tumors, 1,202 carcinomas of the lip, 32 ossifying fibromas, 17 myoblastomas, 59 neurofibromas, and 450 leucoplakias (all oral locations). With such a wealth of material many studies are possible, and the Institute is open to any reputable practitioner who might desire to spend his time in this way. For those who are unable to take advantage of this opportunity, the Registry has provided other facilities to aid the dental practitioner in perfecting his knowledge of pathology, such as the loan study sets, the *Atlas of Dental and Oral Pathology*, and lantern slides.

These aids are available either through the American Dental Association or the Director of the Army Institute of Pathology.

The loan study sets are kept current by the addition of new examples of pathologic material or the replacement of slides by better examples of the same type of case. The *Atlas of Dental and Oral Pathology* has had three revisions to date, and in view of the new cases that have been received, a fourth revision is contemplated in the near future.

The lantern slides and Kodachrome slides cover many aspects of oral diseases, and the lantern slides include both microscopic and macroscopic views of the pathologic condition. The Institute has prepared a numerical list of these slides which is available to interested individuals, in order that they may know what is available for loan.

To have a fundamental understanding of the etiology of disease, a thorough study of the basic conditions and reactions of tissue should be undertaken by all persons attempting to treat diseases. Many dental practitioners are so concerned with the technique of the elimination of diseased teeth and subsequent restorations that they have lost sight of causes or etiology of the condition. The study of pathology in relation to the problem of oral disease and disfunction is of paramount importance. Only a very small percentage of prac-

titioners realize this fact, or those who may realize do not have the necessary basic training or understanding. It is unfortunate that this is the case. It is to be hoped that the facilities offered at the Army Institute of Pathology will be utilized by the dental profession at large to increase the knowledge of dental pathology.

The cooperation of all dental practitioners and their medical conferees who deal with diseases of the oral cavity will make this centralized Registry of Dental and Oral Pathology a source of valuable information from which it may be possible to solve the etiology of some oral diseases.

AN EVALUATION OF THE BIOPSY AS A DIAGNOSTIC AID

JOSEPH L. BERNIER, D.D.S., M.S.*

IN THE clinical interpretation of oral lesions the use of histologic analysis cannot be overemphasized. Since it is not possible to interpret neoplastic and inflammatory lesions properly on the history and clinical appearance alone, biopsy is rapidly becoming a universal diagnostic aid. The files of the Registry of Dental and Oral Pathology contain many cases which were not clearly understood by the clinician, as evidenced by the histologic findings. This is unfortunate since proper analysis of any pathologic process, even though it be basically innocent, is essential in order to rationalize treatment properly. The number of cases in our files in which malignancy has been proved by use of the biopsy further emphasizes the merit of such a procedure in diagnosis. It has often been stated that biopsy is a dangerous procedure; however, these questionable dangers must be very carefully weighed against the valuable information often disclosed. Liljinerantz, as quoted by Kilduff,[1] summarized this by saying, "Whatever the theoretic dangers of biopsy may be, the dangers of attempting radical treatment or abandoning a patient to a hopeless prognosis without tissue proof are far more serious."

It should not be inferred that tissue analysis alone can provide a correct diagnosis in every instance. While occasionally the results obtained are quite dramatic, it is essential that sound clinical judgment on the part of the clinician be employed. While it is often possible to estimate the character of a lesion by the behavior and appearance of the cells, their deviation from normal with respect to staining, and their relation to normal tissue, it is of greatest importance to understand that such information alone cannot provide the complete answer in every instance. The effect of radiation, especially repeated doses, may definitely complicate the problem of microscopic diagnosis. It is important, therefore, that the clinician be aware of this problem in interpreting the report from the pathologist. Further, the location of a lesion may often influence the decision of the clinician regarding the taking of a biopsy. The inaccessibility of deep lesions of bone may cause the procedure to become a major operation, which in certain cases is contraindicated. The decision must be made for each individual case.

As an example of how the biopsy may often assist in the establishment of a correct diagnosis, several cases from the files of the Registry of Dental and Oral Pathology will be reviewed and discussed. In each instance the clinical impression varied from the diagnosis submitted following histologic examination.

*Secretary and Pathologist to the Registry of Dental and Oral Pathology of the American Dental Association, Army Institute of Pathology, Washington, D. C.

Case 1 (A.I.P. Acc. 168756).—

A 16-year-old white boy at routine examination by the school physician was found to have a swelling on the right side of the neck, which had gradually increased in size over the previous several months (Fig. 1). There was no history of discomfort at any time. At operation the mass removed was egg-shaped and extended from the angle of the mandible on the right side to the anterior margin of the sternomastoid muscle and forward to within 1 inch of the midline. The tumor was firm and cystic. There was no opening visible either externally or within the pharynx. The laboratory findings were essentially negative. The clinical impression was mixed tumor.



Fig. 1.—Case 1. Lateral swelling of neck suggesting mixed tumor. The microscopic diagnosis was sclerosing hemangioma.

Microscopic Examination.—The tumor was composed of syncytial masses of deeply staining cells with clear-cut nuclei and prominent nucleoli. The cytoplasm was scant and, where the cells were closely packed, blended with the interstitial fibrous stroma (Fig. 2). The tumor cells had arranged themselves into strands and loops producing numerous vascular channels, some of which contained red blood cells. In the more compact areas the cells were clumped together, suggesting an abortive attempt at capillary budding (Fig. 3). Mitotic figures were occasionally noted. The quality of the cells, as manifested by their size, shape, and staining characteristics, suggested a rapidly growing tumor. In the material submitted the tumor did not appear to have been completely removed.

Diagnosis.—Sclerosing hemangioma.

Comment.—Differential diagnosis of swellings of the neck presents many problems for the clinician. While it is true that in this case a positive diagnosis was not submitted by the operator, still the clinical impression varied from the histologic interpretation. In this instance, through the medium of the biopsy the clinician not only was provided with a correct diagnosis, but was given valuable information regarding the inadequacy of removal. The need for

correctly analyzing benign tumors need not be discussed in detail. Sclerosing hemangiomas, like mixed tumors, may be locally invasive, so perhaps the correct diagnosis in this instance might not have influenced the treatment. The fact that the true nature of the process was determined, however, is of major importance to both the clinician and the patient.

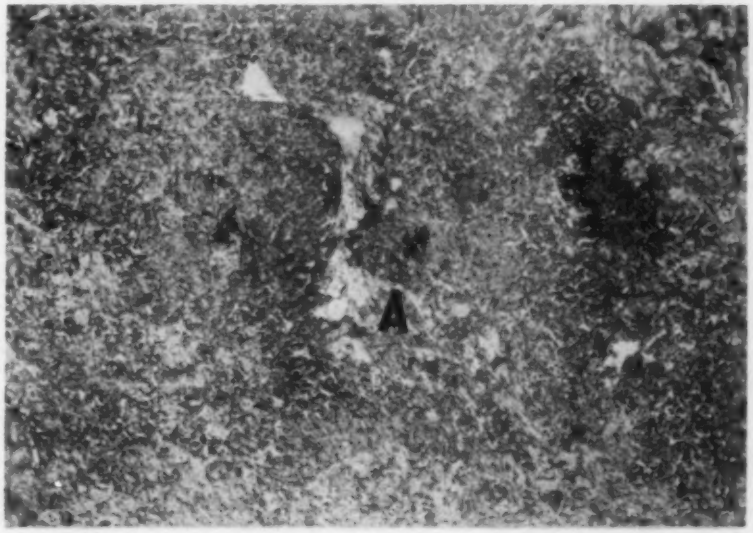


Fig. 2.—Case 1. Low power of tumor in Fig. 1. The intense cellularity is a prominent finding. Note the larger vascular spaces at *A*.

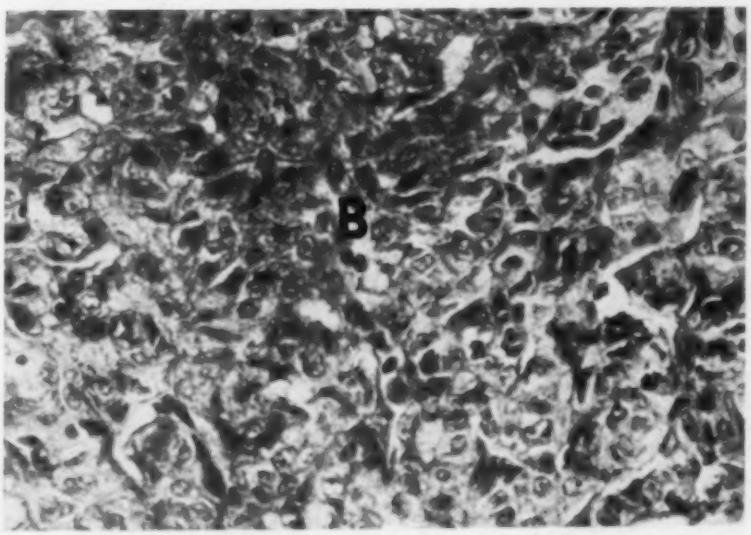


Fig. 3.—Case 1. Higher power of Fig. 2. The character of the cells is shown clearly. There is a suggestion of capillary budding at *B*.

Case 2 (A.I.P. Acc. 165324).—

The patient, a 40-year-old white man, sought relief because of a progressively enlarging swelling on the right side of the lower lip which had caused a slight speech impediment (Fig. 4). The swelling was first noted in 1935 following injury to the lip, the exact nature of which was not disclosed. The clinical diagnosis was retention cyst.



Fig. 4.—Case 2. Unilateral swelling of the lip diagnosed clinically as a retention cyst. This later proved to be a leiomyoma.

Microscopic Examination.—There was a roughly nodular tumor which contained several large vascular channels, some of which were partially filled with red blood cells (Fig. 5). The tumor was composed of homogeneous cells exhibiting prominent nuclear characteristics and abundant cytoplasm (Fig. 6). There was a moderate variation in cellular morphology, although this appeared to be within normal limits. The nuclei, for the most part, were elongated with rounded ends and outlined by a deeply staining nuclear membrane. Fine cytoplasmic projections extended from either end of the cells. The interstitial fibrils formed radiating strands of fibrous tissue. There was a diffuse sprinkling of hemosiderin pigment, most of which was contained in large phagocytic cells.

Diagnosis.—Leiomyoma, upper lip.

Comment.—A variety of tumors occurs as nodular swellings in this location. Although in this instance the tumor was benign, there was a certain aspect of anaplasia which caused some speculation. For this reason the biopsy analysis was of major importance. The fact that the tumor was fairly well encapsulated, a point which could not have been determined clinically, indicates to some extent what may be expected in the future from such a lesion. There was no indication that the tumor would have metastasized.

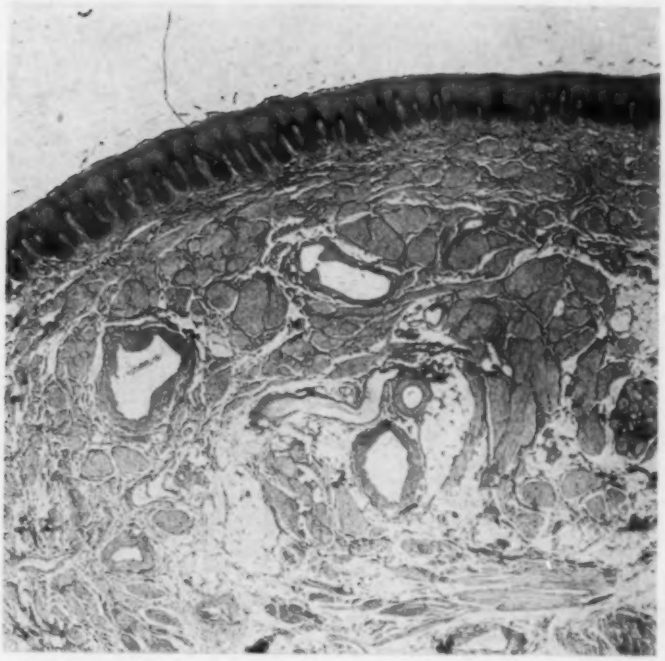


Fig. 5.—Case 2. Leiomyoma of lip. Note bundles of tumor muscle tissue and the vascular feature.

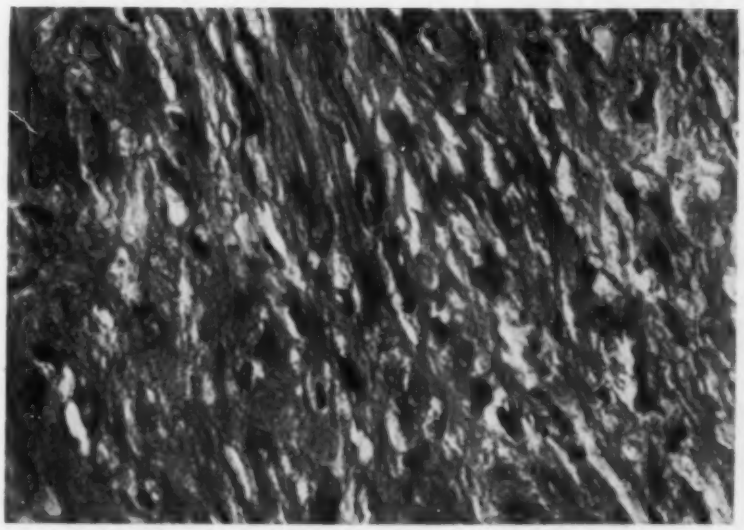


Fig. 6.—Case 2. Higher power of case shown in Fig. 4. The plump rounded nuclei suggest muscle tissue. Masson stains revealed absence of fibrous tissue.

Case 3 (A.I.P. Acc. 168949).—

A 39-year-old Japanese prisoner of war had experienced several episodes of epistaxis. Clinical examination revealed a soft friable mass completely occluding the right nostril. X-ray revealed a cloudy right antrum possibly infiltrated with tumor tissue. No history of previous trauma was given. The clinical diagnosis was "vascular tumor."

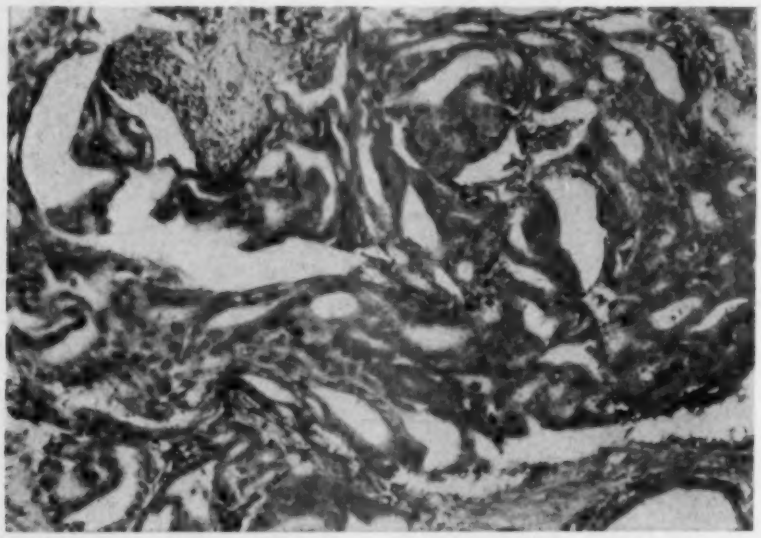


Fig. 7.—Case 3. Lesion from nose of Japanese prisoner of war. Note adenomatous character of the epithelium.

Microscopic Examination.—The irregular section of tumor tissue showed marked superficial ulceration. The substance of the lesion contained irregular islands of epithelium, the cells of which were fairly uniform in size, shape, and staining characteristics (Fig. 7). The epithelial elements tended to assume an acinar configuration, the lumina thus formed containing a shreddy mucinous detritus. Many modified goblet cells were seen in the flattened tubular-like epithelium. The interstitial fibrous connective tissue was but moderately collagenous and was spottily sprinkled with small lymphocytic elements, together with occasional plasma cells and large mononuclear phagocytes.

Diagnosis.—Mucoepidermoid tumor of Bowman's glands.

Comment.—Clinically the true nature of a lesion such as this would tend to be obscure. Only by histologic analysis can one not only determine the structure of the tumor but, as in this instance, suggest a possible point of origin. Of particular interest is the similarity of this lesion to the mucoepidermoid tumors commonly associated with the salivary glands. It is our feeling that histogenetically this tumor would fall within that group. It would appear that this lesion arose from the tubuloalveolar glands of Bowman in the olfactory area of the nasal mucosa. Exhibiting, as it does, some anaplasia, this tumor must be watched carefully for recurrence. Although some of this information might have been obtained by clinical observation, confirmation by the pathologist adds weight to the suggested prognosis. We do not feel that the tumor is malignant, although recurrence, and even local invasion, is a distinct possibility.

Case 4 (A.I.P. Acc. 169023).—

The patient, a 19-year-old white soldier, inadvertently bit his tongue, approximately one year before. A small lesion, with the characteristics of a papilloma, began to grow in the injured site immediately. During the last three months it rapidly increased in size, but no pain or bleeding was associated with its activity. The tumor did not become ulcerated. Removal was accomplished by surgery. The clinical diagnosis was papilloma.

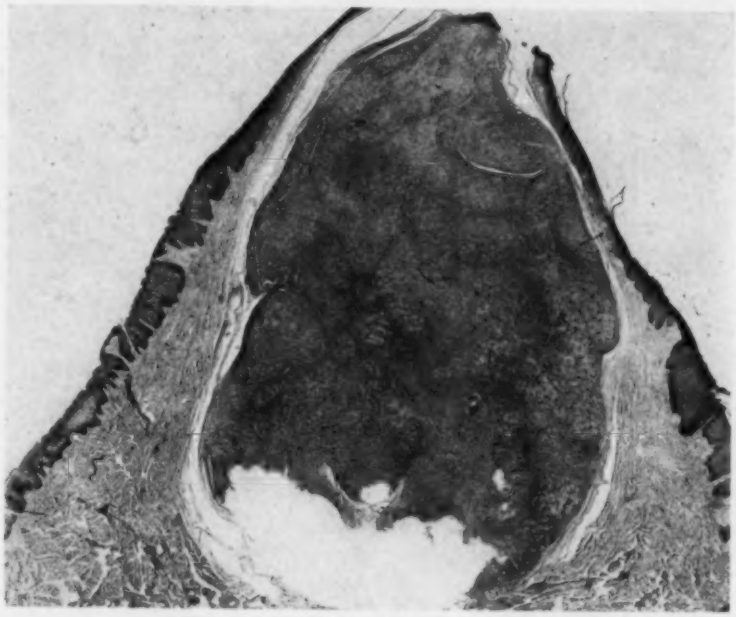


Fig. 8.—Case 4. Tumor from tongue, diagnosed clinically as a papilloma. Biopsy diagnosis was neurofibroma.

Microscopic Examination.—The tumor was composed of fairly well-demarcated lobules of connective tissue cells containing small vesicular hyperchromatic nuclei (Fig. 8). The outer surface was covered by a moderately hyperplastic stratified squamous epithelium exhibiting some spongiosis and parakeratosis. Palisading of the nuclei was a prominent finding (Fig. 9). There was a peculiar whorling of tumor cells which, in some instances, produced circumscribed areas of cytoplasmic condensation suggesting Verocay bodies (Fig. 10). The cells were, for the most part, uniform in size and shape, although occasional normal mitotic figures could be seen. The tumor extended to the base of the specimen submitted, suggesting inadequate removal.

Diagnosis.—Neurofibroma, tongue.

Comment.—This tumor was of considerable interest because of its nature and difficulty of accurate classification. While its clinical appearance suggested a papilloma, the histologic analysis revealed a more specific entity, together with the fact that it had been inadequately removed. Under such conditions, of course, recurrence is a likely possibility. Any attempt to classify neurogenic fibromas on the basis of etiology leads to much confusion. It is possible, in

Case 3 (A.I.P. Acc. 168949).—

A 39-year-old Japanese prisoner of war had experienced several episodes of epistaxis. Clinical examination revealed a soft friable mass completely occluding the right nostril. X-ray revealed a cloudy right antrum possibly infiltrated with tumor tissue. No history of previous trauma was given. The clinical diagnosis was "vascular tumor."

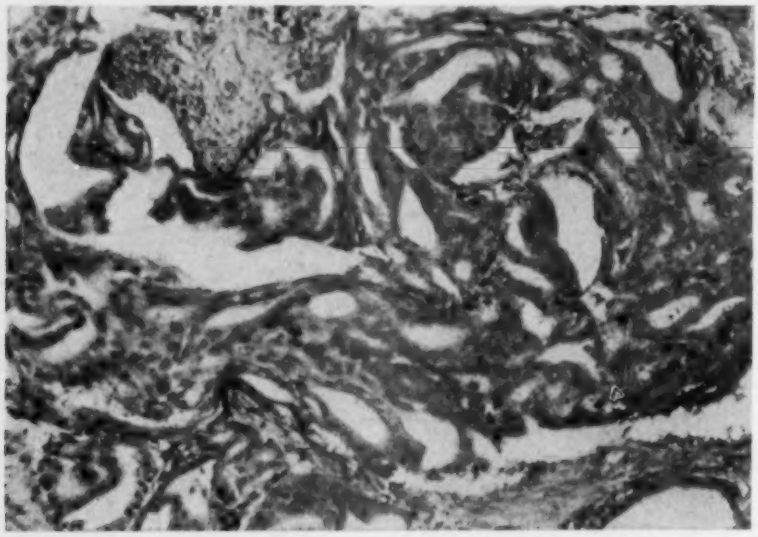


Fig. 7.—Case 3. Lesion from nose of Japanese prisoner of war. Note adenomatous character of the epithelium.

Microscopic Examination.—The irregular section of tumor tissue showed marked superficial ulceration. The substance of the lesion contained irregular islands of epithelium, the cells of which were fairly uniform in size, shape, and staining characteristics (Fig. 7). The epithelial elements tended to assume an acinar configuration, the lumina thus formed containing a shreddy mucinous detritus. Many modified goblet cells were seen in the flattened tubular-like epithelium. The interstitial fibrous connective tissue was but moderately collagenous and was spottily sprinkled with small lymphocytic elements, together with occasional plasma cells and large mononuclear phagocytes.

Diagnosis.—Mucoepidermoid tumor of Bowman's glands.

Comment.—Clinically the true nature of a lesion such as this would tend to be obscure. Only by histologic analysis can one not only determine the structure of the tumor but, as in this instance, suggest a possible point of origin. Of particular interest is the similarity of this lesion to the mucoepidermoid tumors commonly associated with the salivary glands. It is our feeling that histogenetically this tumor would fall within that group. It would appear that this lesion arose from the tubuloalveolar glands of Bowman in the olfactory area of the nasal mucosa. Exhibiting, as it does, some anaplasia, this tumor must be watched carefully for recurrence. Although some of this information might have been obtained by clinical observation, confirmation by the pathologist adds weight to the suggested prognosis. We do not feel that the tumor is malignant, although recurrence, and even local invasion, is a distinct possibility.

Case 4 (A.I.P. Acc. 169023).—

The patient, a 19-year-old white soldier, inadvertently bit his tongue, approximately one year before. A small lesion, with the characteristics of a papilloma, began to grow in the injured site immediately. During the last three months it rapidly increased in size, but no pain or bleeding was associated with its activity. The tumor did not become ulcerated. Removal was accomplished by surgery. The clinical diagnosis was papilloma.

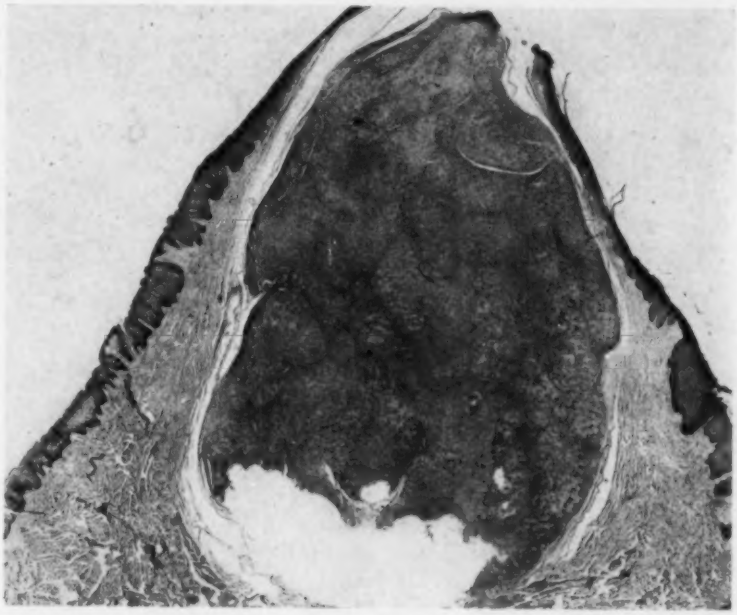


Fig. 8.—Case 4. Tumor from tongue, diagnosed clinically as a papilloma. Biopsy diagnosis was neurofibroma.

Microscopic Examination.—The tumor was composed of fairly well-demarcated lobules of connective tissue cells containing small vesicular hyperchromatic nuclei (Fig. 8). The outer surface was covered by a moderately hyperplastic stratified squamous epithelium exhibiting some spongiosis and parakeratosis. Palisading of the nuclei was a prominent finding (Fig. 9). There was a peculiar whorling of tumor cells which, in some instances, produced circumscribed areas of cytoplasmic condensation suggesting Verocay bodies (Fig. 10). The cells were, for the most part, uniform in size and shape, although occasional normal mitotic figures could be seen. The tumor extended to the base of the specimen submitted, suggesting inadequate removal.

Diagnosis.—Neurofibroma, tongue.

Comment.—This tumor was of considerable interest because of its nature and difficulty of accurate classification. While its clinical appearance suggested a papilloma, the histologic analysis revealed a more specific entity, together with the fact that it had been inadequately removed. Under such conditions, of course, recurrence is a likely possibility. Any attempt to classify neurogenic fibromas on the basis of etiology leads to much confusion. It is possible, in

some instances, to suggest origin from the nerve sheath to the exclusion of the Schwann layer, although according to some investigators this is hazardous, at best. Certainly such an insight into the histology of this lesion would not be possible on clinical analysis alone.

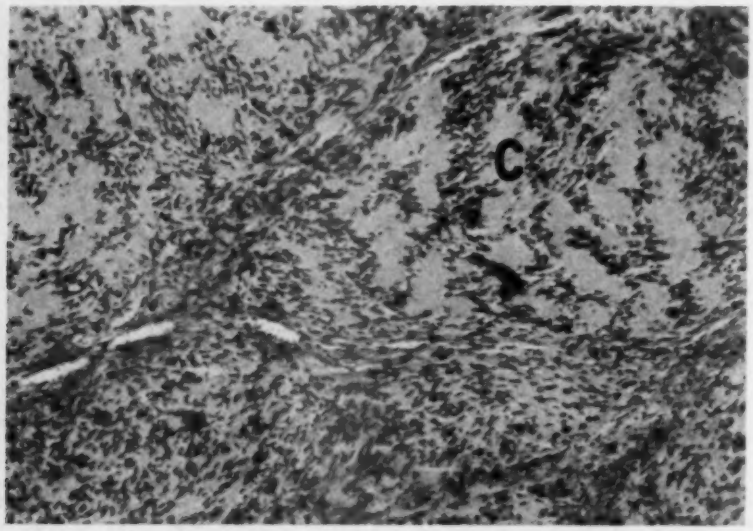


Fig. 9.—Case 4. Higher power of Fig. 8, showing palisading of nuclei at *C*.

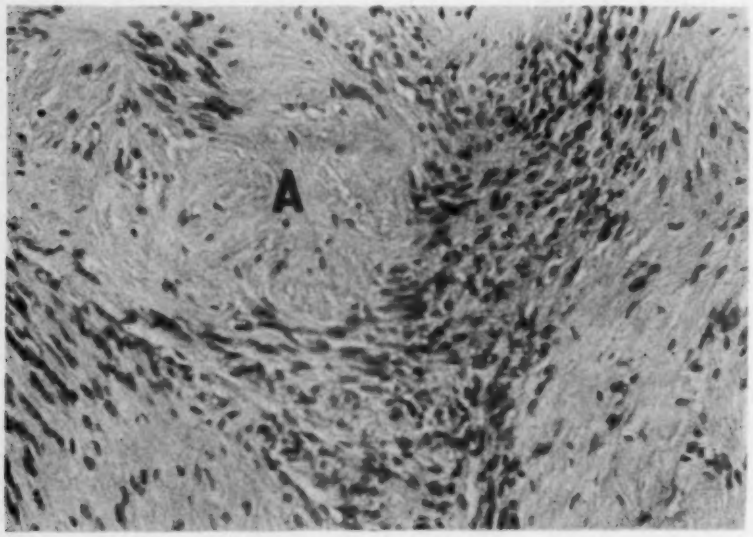


Fig. 10.—Case 4. Higher power of Fig. 9. Note Verocay bodies at *A*.

Case 5 (A.I.P. Acc. No. 168871).—

A 26-year-old white man had a small nodular tumor on the buccal surface of the maxillary mucosa between the upper left central and lateral incisor teeth. This lesion was first noticed by the patient five months previously. It had steadily increased in size, but at no time were there any symptoms. There

was no history of associated trauma. At time of removal, the tumor was found to be rather loosely attached to the underlying connective tissue elements. The mucosa surrounding the lesion was essentially normal. No periodontitis was evident. The clinical diagnosis was "epulis."

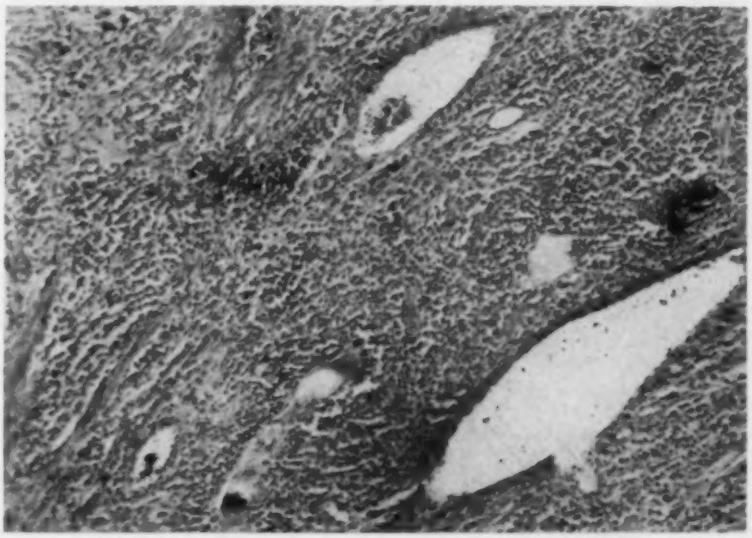


Fig. 11.—Case 5. Low power of intraoral tumor diagnosed clinically as "epulis." Note large vascular spaces and abundance of cells.

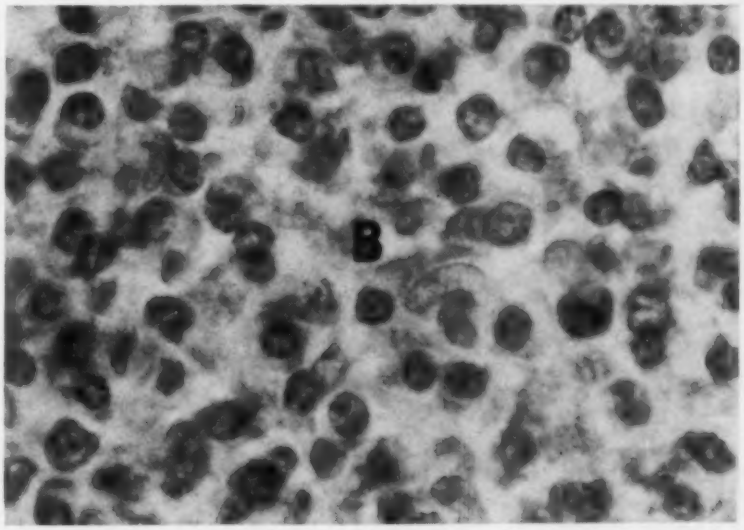


Fig. 12.—Case 5. The "wheel" or "spoke" nuclei characteristic of the plasma cell are clearly shown at *B*. Diagnosis was plasmocytoma.

Microscopic Examination.—A roughly spherical tumor mass was covered by markedly hyperplastic stratified squamous epithelium exhibiting a very slight parakeratosis. There was some hydropic degeneration in the granular layer. The tumor was composed of very dense collections of deeply staining

uniform cells containing radiating nuclear inclusions. These were typical plasma cells (Figs. 11 and 12). Moderately collagenous trabeculae of fibrous tissue extended freely between these cell collections. Only occasional small lymphocytic elements were in evidence. Vascularity was moderate, some of the vessels being dilated and partially filled with red blood cells. There was a sparse sprinkling of eosinophilic leucocytes.

Diagnosis.—Plasmocytoma, maxilla.

Comment.—Often plasma cells may be present in the gingiva in such abundance that neoplasia is simulated. True plasmocytomas, however, should be differentiated from lesions of an inflammatory nature. The former includes tumors so common in the conjunctiva and the mucosa of the upper respiratory tract. They are usually single and benign. An occasional one, particularly when located in the nasopharynx, has been known to metastasize. These tumors are akin to the so-called multiple myelomas which occur in bone. The use of the term epulis, in this case, is another example of an inadequate clinical designation for a tumor of definite histologic pattern. It is quite unlikely that a clinical diagnosis of plasmocytoma could be rendered on appearance alone. This, then, further emphasizes the necessity for accompanying tissue analysis to determine the true nature of the process. As in the previous cases, removal of this lesion is inadequate, as estimated from the tissue submitted. It is most important that the clinician have this information in order that he may rationalize further treatment properly.

Case 6 (A.I.P. Acc. 153856).—

A 25-year-old white man injured the mucosa of his lower lip while shaving. During the next four months the lesion continued to heal and then ulcerated. A hornlike swelling eventually appeared at the site of injury, which continued to grow until it became approximately the size of a green pea (Fig. 13). There was a thick roughened projection, grayish-white in color, which was rough to digital exploration. The clinical diagnosis was papilloma. Under local anesthesia the tumor was removed and submitted in toto for histologic analysis.

Microscopic Examination.—A small segment of lip mucosa, the epithelium of which was markedly acanthotic was seen. There was considerable parakeratosis with an accompanying hyperplasia of the stratum granulosum. Some spongiosis was evident. The elongated rete pegs extended into the reticular layer of the corium and at the edges of the lesion tended to fold inward, suggesting a verrucoid character (Fig. 14). The corium was moderately vascular and sparsely sprinkled with chronic inflammatory elements. There were several isolated bundles of striated muscle. There was a very slight dyskeratosis in evidence, although for the most part the cells were uniform morphologically. There was a sparse chronic inflammatory exudate in the corium.

Diagnosis.—Verrucal type of leucoplakia.

Comment.—The proper understanding of lesions affecting the lips is of extreme importance to the clinician. Tumors affecting the upper lip are much

less prone to exhibit malignant changes. Those affecting the lower lip, on the other hand, from even the slightest trauma, may exhibit marked malignant change. In the classification of lip tumors the Registry recognizes the verrucal type of leucoplakia as a definite entity, being a counterpart of senile keratosis of skin. It is our feeling that dyskeratosis is a necessary part of the histologic picture of these lesions and is sufficient to place them in the category of precancerous. In those lesions exhibiting marked epithelial activity without severe dyskeratosis, the term pseudoepitheliomatous hyperplasia may be considered.



Fig. 13.—Case 6. Lesion of lip diagnosed clinically as a papilloma. The lesion proved to be a verrucal form of leucoplakia.

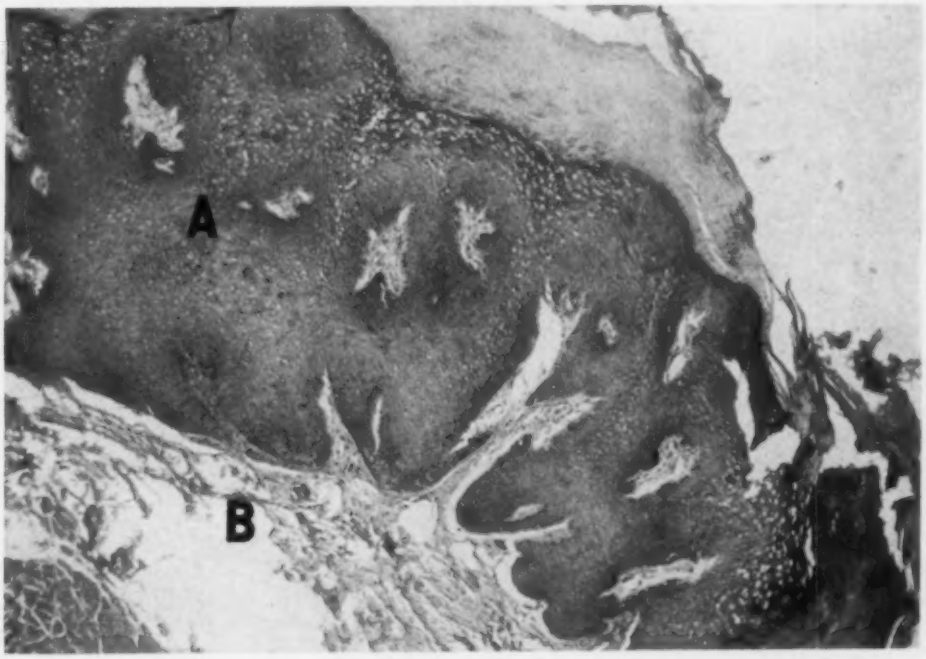


Fig. 14.—Case 6. Microscopic section of lesion shown in Fig. 13. Note acanthosis of epithelium (*A*) and absence of inflammatory exudate in corium (*B*).

This, of course, infers an excessive hyperplasia of the overlying mucosa deeply penetrating the underlying corium. No degree of clinical skill will enable the clinician to classify such lesions on the basis of appearance only. It is essential that the pathologist be consulted in order to rationalize treatment and to estimate their prognosis.

SUMMARY

It has not been the purpose of this discussion to deal intimately with the histologic nature of the cases reported; rather, an attempt has been made to show the necessity for histologic examination, in order to catalogue tumors and diseases properly. Perhaps the impression of overemphasis on the biopsy is obtained from such a discussion. If so, it should be again emphasized that the biopsy cannot replace sound clinical judgment or any of the many other diagnostic aids. It can and does, however, often contribute much concrete information as to the exact character of the tumor or disease in question. This information, however, must be properly correlated by the clinician in order to evaluate truly the process in question. The many erroneous beliefs concerning the dangers of biopsy, particularly in suspected malignant lesions, must be approached critically. Being essentially a simple process, one can estimate these dangers in comparison to an inadequate clinical interpretation. This, then, is a plea for the more universal employment of the biopsy as a diagnostic procedure, not only by the exodontist and oral surgeon, but by the general dentist, as well. It is important that whoever sees lesions of the mouth should fully understand their potentialities, in order to increase his ability to provide an adequate service for the patient.

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THE HISTOGENESIS OF THE CEMENTOMA

Report of 15 Cases

Joseph L. Bernier, D.D.S., M.S.,*† and Henry C. Thompson, D.D.S.‡

THE histogenesis of the cementoma, one of the most common of the odontomas occurring in pure form, is not too well understood. Most authors have described the basic process as a continued deposition of cementum about a central nidus, which may be either a focal center of calcification or an isolated cementicle. Like many tumors arising from a tissue which is potentially capable of producing calcified material, it is initially a soft tissue lesion. The earliest stage, when the cellular stroma is prominent, is often referred to as the cementoblastic period. Later in the terminal stages of growth, when calcified tissue is present in larger quantities, the term cementoma is employed. Thoma[1] has referred to the earlier stages of development as fibromatous or osteolytic. The term osteolytic is not descriptive in every instance and, therefore, may not be too significant. Initially, there is no appreciable degree of cementum formation, the periphery of the mass being radiolucent, hence the basis for the term fibromatous. The intermediate stage he has referred to as the cementoblastic period, or cementum-forming stage, at which time the tumor begins to produce cementum, either as cementicles or in a trabecular pattern (Fig. 1). Terminally, the tumor assumes an inactive growth status and is referred to as the cementoma. Further growth after this period is inconsequential. The fibrous connective tissue forming the matrix of the tumor is essentially similar to that found in the periodontal membrane, in that it is potentially capable of producing both bone and cementum. The impetus necessary to call forth the production of either of these tissues is a matter of conjecture. Continued observance of the reaction to inflammation of tissue potentially capable of producing a calcified material suggests that in some instances this may be one of the stimuli responsible for such a proliferation. In the early stages of activity, granulomas, radicular cysts, and rarefaction due to trauma may simulate this process because of a similar roentgenographic appearance. Because of this, Stafne[2] feels that many mistaken diagnoses have been made. This is undoubtedly true. Stafne[3] has referred to the initial development of this lesion as the periapical fibroma. He has suggested that continued development may follow one of three courses: (1) most of the fibrous tissue may be converted to cementum; (2) it may remain in the transitional or cementoblastic stage; or (3) it may be replaced by bone. This

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latter possibility is not common. Gottlieb[4] has suggested that the nidus for the formation of cementicle in the growing tumor may be degenerated epithelial cells from the rests of Malassez. Bauer[5] feels that thrombosed vessels or detached fragments of cementum may also act as the nucleus, although this is somewhat speculative. In those instances where there has been an excessive production of cementum within the tumor, it may be difficult to suggest an accurate clinical diagnosis. Such lesions have occasionally been referred to as calcified granulomas.

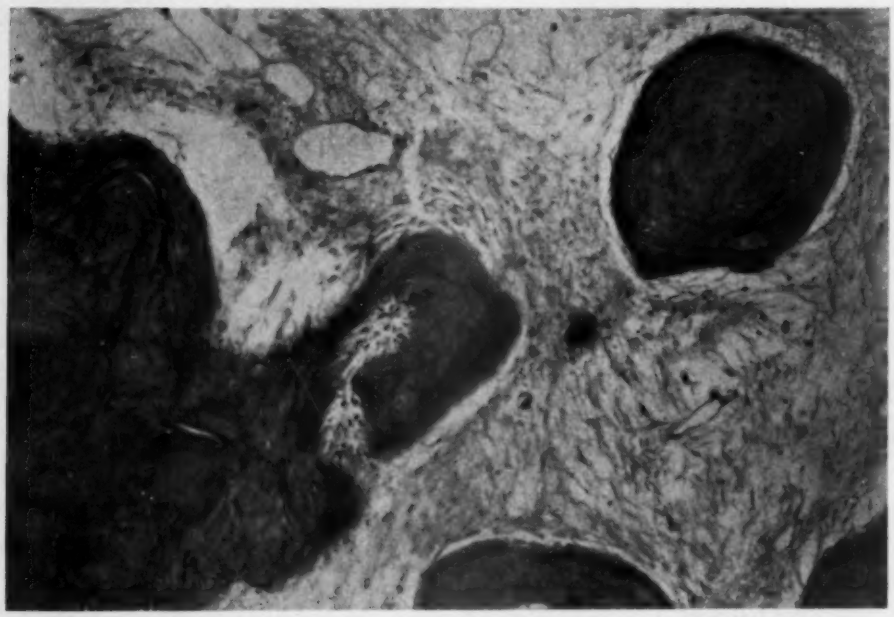


Fig. 1.—Intermediate stage of cementoma. The cementum has been deposited as cementicles. The fibrous stroma is loose and edematous. Note the proliferating fibroblasts.

Terminally, the cementoma may be recognizable roentgenographically as a radiopaque area surrounded by a thin zone of radiolucency. At this time, the calcified portion of the tumor has greatly overgrown the fibrous tissue matrix. The bone surrounding a mature lesion is usually dense, as a result of pressure from the growing tumor. In certain instances, the basic pattern of fused cementicles may be overshadowed by the continued deposition of cementum. Such lesions are occasionally confused histologically with those having a frank trabecular appearance. Stafne[6] reported 35 cases of cementoma following roentgenologic examination of 10,000 consecutive adult patients. Seventy-eight teeth were involved in these lesions. The ratio of the mandibular to the maxillary lesions was 17 to 1, and the age of occurrence ranged from 20 to 62 years of age. Since cementomas usually occur some distance from the apices of adjacent teeth, it is of interest to speculate on their formation in these areas. As a rule, cementicles are found in the periodontal membrane (Fig. 2). However, they may remain behind a drifting tooth, being subsequently seen at some distance from the tooth. Epithelial debris may in a like manner be found some distance

TABLE I. SUMMARY OF REPORTED CASES

	CASES	AVERAGE AGES	SEX M	SEX F	RACE W	RACE N	RATIO OF LESION IN MAX. TO MAND.
Stafne	65	42.5	19	46			1 to 17
Thoma	3	46	1	2	3	0	0 to 3
Bradley	2	43		2	2		0 to 2
Dewey	1	40		1	1		0 to 1
Zegarelli and Ziskin	50	40	0	50	34	33	1 to 7
Our group	15	37.2	7	8	10	5	1 to 23
Total	136				57%	W	
Averages		41.5	1 to 4		43%	N	1 to 17.7



Fig. 2.—Showing cementicles in periodontal membrane. There is a focal accumulation of cementum on the apex of the tooth root.

from the apex and either of these may serve as a nidus for the formation of a cementoma. Zegarelli and Ziskin[7] reported 50 cases of cementomas in females. In their opinion, no systemic disease could be associated with their formation. They did, however, suggest that endocrine or metabolic disturbance may on occasion exist. (See Table I.) Burn, Orten, and Smith[8] produced odontomas in over 60 per cent of albino rats surviving 365 days on a mild vitamin A deficiency. The average daily dose was 0.7 to 0.8 International Units. Case reports, as a rule, aid greatly in the clinical and histologic study of tumors. The following are from the files of the Registry of Dental and Oral Pathology and will be discussed in order to emphasize the more important histologic features of the cementoma.

Case 1 (A.I.P. Acc. No. 131700).—

History.—A 26-year-old white woman had a small cavity filled in the lower left central incisor in 1939. In 1940 she struck her chin and fractured the right



Fig. 3.—Case 1. Large cementoma which necessitated removal of central and lateral incisor. Note the zone of radiolucency about the tumor.

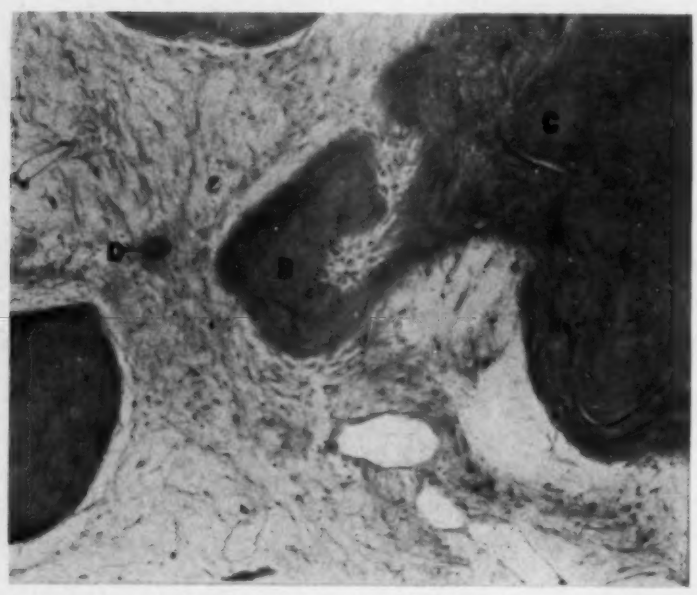


Fig. 4.—Microscopic section of case shown in Fig. 3. There is a small cementicle at *D*. Note the cementoblastic activity at *B*. At *C* previously formed cementicles have fused.

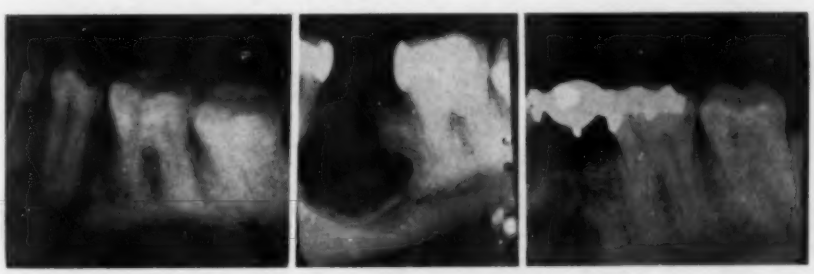
condylar process. She suffered no ill effects at the site of trauma, other than a small laceration. In 1944 the lower left central incisor was refilled. At this time there was some pain from the tooth, which became more severe during the succeeding six months. The roentgenograms revealed a dense and apparently unattached area below the lower incisor tooth (Fig. 3). This mass could be palpated through the lower lip.

Operation.—A spherical mass covered with a thin layer of normal bone was removed through a labial opening. A fibrous membrane was adherent to the spherical mass and to the apices of the lower right and left central incisors. Because of this, these teeth were removed.

Microscopic Examination.—There were several fragments of a cellular fibrous tissue containing numerous fragments of calcified material having the general characteristics of cementum. The fibrous tissue was composed of plump active fibroblasts enmeshing many small vascular channels (Fig. 4). The trabeculae of cementum were, in some areas, bordered by active cementoblasts, some of which were being incorporated in a preliminary cementoid-like matrix. The lacunar characteristics of the embedded cementocytes were strongly suggestive of an abortive type of cementum. The impression of a rapidly growing tumor was suggested by the histologic pattern.

Case 2 (A.I.P. Acc. No. 80340).—

History.—A 32-year-old white woman had the lower second premolar, together with an adherent calcified mass, removed under local anesthesia in 1937 (Fig. 5, *A*). Periodic roentgenograms indicated a recurrence in 1940 (Fig. 5, *B*) and again in 1942 (Fig. 5, *C*). The mass grew larger during the subsequent year.



A. *B.* *C.*

Fig. 5.—Case 2. The initial lesion is shown in *A*. The second recurrence is shown in *B*, and the third recurrence in *C*.

Operation.—An intraoral incision through the buccal plate of the mandible was made. A spherical mass, approximately 7 mm. in diameter, was removed. There was a thin membrane lining the cavity.

Microscopic Examination.—The tumor was composed of many irregular trabeculae of cementum containing distorted incremental lines (Fig. 6). The interstitial fibrous stroma was moderately abundant and contained plump actively growing fibroblasts (Fig. 7). Throughout the section the trabeculae of

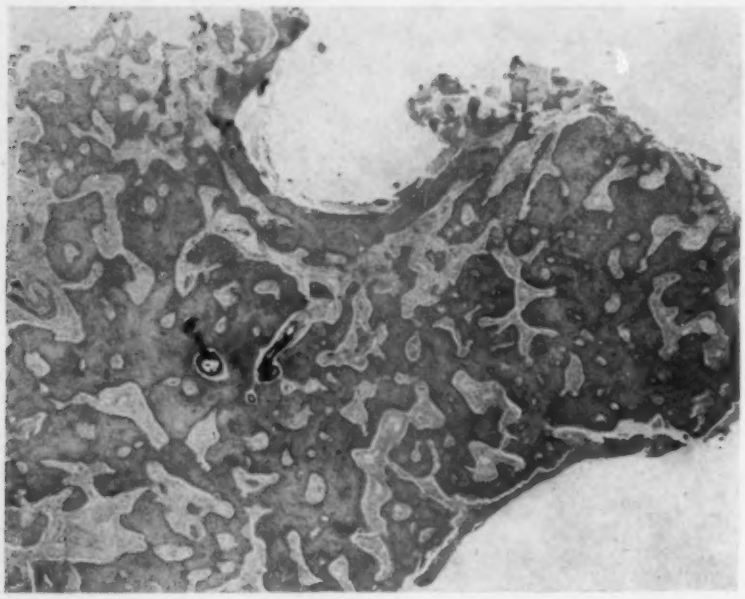


Fig. 6.—Section of tumor in Fig. 5. There is considerable cementum arranged in a trabecular pattern. The stroma is moderate and vascular.



Fig. 7.—Higher power of Fig. 6. The character of the cementum is clearly shown. Each trabecula is outlined with actively proliferating cementoblasts.

cementum were bordered by cementoid material which contained actively proliferating cementoblasts. There was a suggestion of edema throughout the fibrous stroma, although no inflammatory elements were in evidence. At one corner of the specimen the fibrous tissue was quite abundant. This was the rapidly growing portion of the tumor, as evidenced by the many minute islands of cementoid tissue.

Case 3 (A.I.P. Acc. No. 121256).—

History.—A 38-year-old white man, seen in 1944, had a radiolucent area in the region of the lower left first molar. This appeared to be a cystlike cavity with a spherical calcified mass in the center. The lower left second premolar and first molar had been extracted in 1939 with some resultant postoperative difficulty. In 1940 the patient received a blow to the jaw which did not produce any significant symptoms.

Operation.—A hard bonelike nodule approximately 10 mm. in diameter was removed from the cavity in the left mandible.

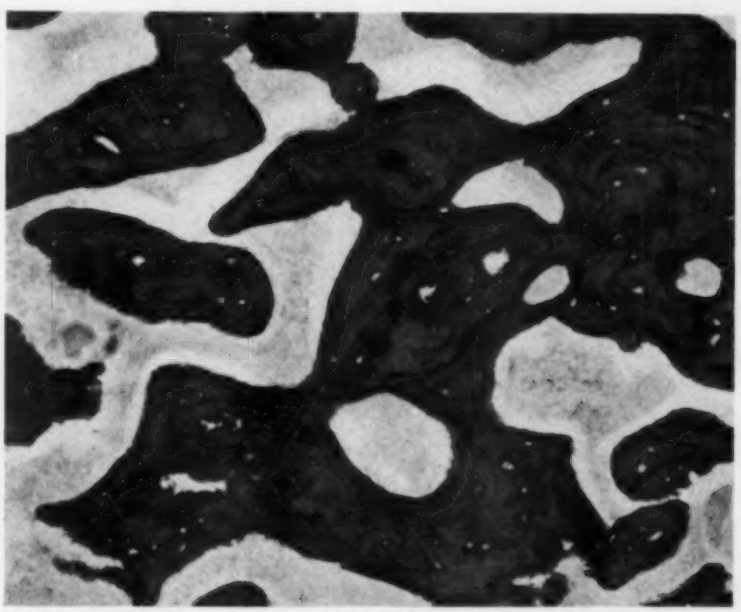


Fig. 8.—Section of lesion in Case 3. There is an irregularly formed cementum. The stroma is badly autolyzed. In other areas the fibrous tissue was active.

Microscopic Examination.—There were several fragments of tumor tissue, the largest of which appeared to be a solid mass of irregular cementum containing occasional islets of collagenous fibrous connective tissue (Fig. 8). The remaining sections contained an abundant fibrous stroma in which were enmeshed many irregular trabeculae of poorly formed cementum. The fibrous tissue matrix appeared excessively autolyzed. There were only occasional cementoblasts noted. It did not appear that this was a rapidly growing tumor. Vascularity was moderate.

STATISTICS

Fifteen cases of cementomas are included in this report. As a preliminary study, all of our odontomas were reviewed with the purpose of eliminating any cementoma which might be considered of the mixed type of odontoma. The average age of occurrence was 37 years, the youngest patient being 23 years of age and the oldest 57 years of age. Eight cases (54 per cent) were in females and 7 (46 per cent) in males. The average duration of these lesions, on the basis of available information, was five years. This figure should be accepted with some misgivings, inasmuch as it is based on the questionable recollection of the patient. It does, however, suggest that the cementoma is essentially a slow-growing tumor, a fact emphasized by many authors. According to most investigators, the mandible is more frequently affected than the maxilla. This is borne out in our group, inasmuch as but one maxillary tooth was involved, as compared with 22 mandibular teeth. Twelve, or 54.5 per cent, of the lower teeth were in the anterior portion of the mandible, while 10, or 45.5 per cent, were located in the posterior mandibular region. Five patients (33.3 per cent) reported pain in the teeth involved. This is important, in that, although the tumor itself is painless, such secondary manifestations may be the means of recognition.

Histologically, 10, or 66.6 per cent, of the growths were composed of irregular trabeculae, while 5, or 33.4 per cent, showed definite evidence of cementicle formation. Six, or 40 per cent, were attached to the teeth immediately adjacent. Likewise, 6, or 40 per cent, of the lesions were surrounded by a fibrous capsule, from which they shelled out like peas. This would indicate that these tumors were still in the formative stage. While all of the lesions exhibited some measure of encapsulation, only in this group was this a significant finding. All of these data are shown in Table II.

TABLE II. SUMMARIZED DATA

AGE (YEARS)	RACE	SEX	DURATION (YEARS)	APEX AREA		FORM	ATTACHED TO ROOT	SYMPTOMS
				MAXILLA	MANDIBLE			
23	N	M	4		R 9	Trabeculated	No	Pain
25	W	M			R16	Trabeculated	No	
26	W	F	4		R9, L9	Trabeculated	Yes	Pain
26	W	M			R9, 10, 11, 12, L9, 10, 11, 12	Trabeculated	Yes	Pain
32	W	F	5		R13	Trabeculated	No	
34	W	M			L14	Trabeculated	Yes	
35	W	M			R14	Cementicles	Yes	
38	W	M	5		L14	Trabeculated	No	Pain
40	W	F			R14	Trabeculated		
41	W	F	6		L14	Trabeculated	Yes	
43	W	M		R8		Trabeculated	Yes	
44	N	F			L14	Cementicles		
44	N	F			R10	Cementicles		
45	N	F			L14	Cementicles		Pain
57	N	F			R14	Cementicles	No	
Total	10 W 5 N	7 M 8 F		1	22	10 trabeculated, 5 cementicles	6 attached	5 pain
Average 37			5					

DISCUSSION

Productive fibrosis in the periapical region, seemingly a common manifestation, may be the initial reaction in the formation of cementomas. Without entering into any discussion concerning the fine line of division between hyperplasia and neoplasia, it seems fair to assume that this reaction might be considered as an early manifestation of tumor formation. At least, one should accept this terminology if the cementoma is to be considered a true neoplasm. Fig. 9 shows such an increased width in the periodontal membrane with beginning collagenation of the fibrous connective tissue. Perhaps at this stage one is forced to accede to the suggestion of hyperplasia, rather than neoplasia.

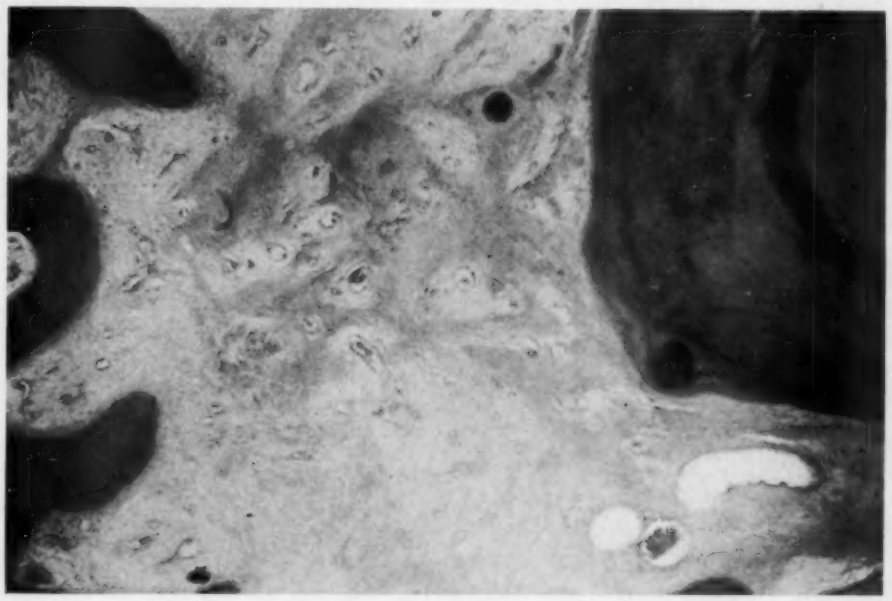


Fig. 9.—Early increase in fibrous tissue in periodontal membrane. There is some increase in collagen.

Such a differentiation from a practical standpoint is largely academic, because in the face of this type of proliferation the periodontal membrane, which possesses the inherent capabilities of producing both bone and cementum, may react with the subsequent formation of a calcified tissue. This, then, is the initial phase of cementoma formation. On the basis of the histology alone, one must consider this reaction as basically similar to the peripheral fibrous tissue lesions associated with the periodontal membrane, inaccurately referred to as epulides. Indeed, it seems likely that those bone manifestations occurring in the maxilla and mandible, commonly referred to as ossifying fibroma, fibrous osteoma, or fibrous dysplasia of bone, may be essentially similar in their earlier stages. Transition of the normal hemopoietic tissue to a fibrous matrix may be the result of this invading proliferation of the periodontal membrane. In certain instances this reaction may be complicated by the presence of epithelial rests which go on to hyperplasia, as shown in Fig. 10. The presence of epithelial debris and isolated focal centers of calcification in appreciable quantities in the periodontal

membrane may shed some light on the formation of the cementoma. Given a central nidus about which to deposit cementum or bone, fibrous tissue, as found in the periodontal membrane, may well proceed to such an accomplishment. The continued growth of this lesion may eventually place it in the category of a clinically and microscopically significant tumor.

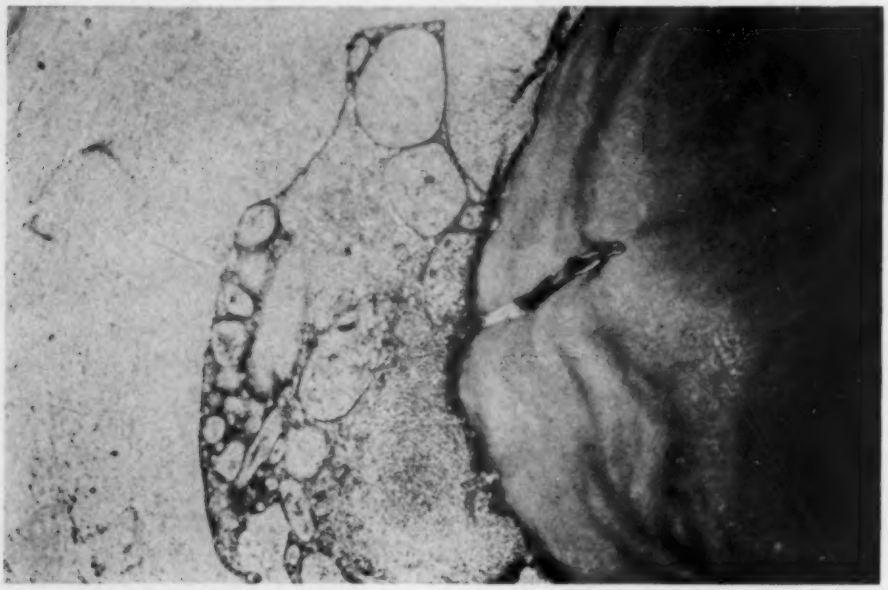


Fig. 10.—Proliferating epithelium at apex of central incisor. Note the chronic inflammatory exudate.

On the basis of such an analysis, it is questionable whether the cementoma can rightfully be considered an odontoma. The association with the perifollicular mesenchymal tissue through the medium of the periodontal membrane is, of course, indisputable. However, one must consider that this tissue, representing the anlage of the periodontal membrane, is not clearly defined and blends freely with the neighboring connective tissue. One need only remember that the periodontal membrane in its normal functional role produces bone, as well as cementum, and that this former activity can hardly be considered a function of the odontogenic apparatus.

While we have recognized in our survey two histologic manifestations of the cementoma, we do not consider these particularly significant. Whether the cementum is deposited about a central nidus or is produced in a trabecular pattern apparently represents merely an alteration in the physical pattern followed by the growing tumor. The essential process is, of course, the activity of the fibrous connective tissue, which is normally capable of producing these calcified tissues. An understanding of peripheral and central fibrous tissue tumors occurring elsewhere in the body is essential if one is properly to rationalize such reactions in the periodontal membrane. The simplest analysis seems to suggest that the periodontal membrane, being a specialized form of periosteum, reacts much as periosteum elsewhere in the human body. Modifica-

tions in tumors arising from this tissue are explained on the basis of its additional ability to produce cementum and the presence of epithelial debris. It does not seem likely that inflammation plays an important role in stimulating the periodontal membrane. While the instance of periodontitis, as compared with periostitis elsewhere in the human body, is considerably greater, still the production of either peripheral or central connective tissue tumors is not in the same ratio. That the cementoma need not necessarily be associated with a normal-functioning periodontal membrane has been observed in our cases. Fig. 11 is an x-ray of a lesion involving the ramus of the mandible and bearing no physical relationship with a functioning periodontal membrane. In this case the lower third molar was extracted before there was clinical evidence of the tumor.



Fig. 11.—Cementoma involving most of ramus. The third molar had been extracted before the tumor was evident.

Fig. 12 illustrated another case of cementoma, one associated with a normal periodontal membrane. In this instance, however, the tumor shows a recurrence. Even so, one must rationalize such continued activity in the absence of any possible periodontal inflammation.

If the border line between the cementoma and other bone-producing lesions affecting the maxilla and mandible is not clearly defined, one then has little justification for assuming that the cementoma is a rare lesion. In many instances it is difficult to identify the calcified tissue in so-called cementomas

as cementum. This aberration, due to the disorganized activity of the cementoblasts and osteoblasts, makes a clear-cut analysis difficult. It seems reasonable that neither clinical nor roentgenographic data alone are sufficient to support a diagnosis of cementoma, in view of the fact that the true nature of the tumor is difficult to recognize, even microscopically.



Fig. 12.—Recurrent cementoma in edentulous mandible.

SUMMARY

Fifteen cases of cementoma are reported and discussed. This material is presented with the suggestion that the reaction of the periodontal membrane in the production of the cementoma may be essentially similar to the process resulting in the formation of ossifying fibromas, fibrous osteomas, and fibrous dysplasia of bone. If this analogy is true, then one is not entirely justified in assuming that the cementoma is a rare tumor. It likewise becomes important, then, to place more stress on the histologic analysis of such lesions if an accurate diagnosis is to be forthcoming.

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INVASION OF PARANASAL SINUSES BY TUMORS OF MENINGEAL ORIGIN

LT. COL. WILLIAM W. HURTEAU, M.C., UNITED STATES ARMY*

WHILE meningiomas occasionally infiltrate through the skull over the convexity of the cranium to present in the overlying scalp or temporal muscle, infiltration of the base of the skull is unusual, and infiltration into the paranasal sinuses is rare.

The histogenesis and nomenclature of these tumors are controversial. Many investigators agree that the type cells of origin are the arachnoidal cell clusters, particularly the arachnoidal mesothelial cells forming the caps of the Pacchionian granulations. However, the nature of these arachnoidal lining cells is in dispute. Mallory (1920), regarding them as undifferentiated potential fibroblasts, proposed the term "arachnoid fibroblastoma" for the tumors. Penfield (1927) endorsed the conception that they are fibroblastic and regarded these tumors as "meningeal fibroblastomas." Bailey and Bucy (1931) objected to classifying all of these tumors as fibroblastomas and recommended a division into nine different types according to structure. In their classification the fibroblastic type was recognized as an uncommon one. Foot (1940) believed the cells of origin to be derived from the neural crests. These and many other divergent opinions as to nomenclature and histogenesis led Cushing (1922) to propose the name "meningioma" to include the entire group, connoting simply that the tumor arises from the meninges.

While the distribution of meningiomas most frequently follows the distribution of the Pacchionian granulations, it is possible that they may arise from arachnoidal cell aggregates anywhere along the arachnoidal membrane and even outside of the intracranial cavity in the optic nerve sheaths. Weed (1920) found from observations on the meninges of cats that the mesothelial cells lining the arachnoid membrane were subject to morphologic variation. These cells not only formed free macrophages but in senescence proliferated to form small, flattened nodules in which calcification occasionally was seen. In addition to these cell aggregates, frank new growths were observed in some of the series, which were so similar to the smaller clusters as to suggest that the neoplasm was the end result of hyperplasia of the arachnoid cell cluster. These neoplasms were typical meningiomas.

Scattered overgrowths of arachnoidal cells are not uncommonly found in man and may well be related to the formation of meningeal tumors. They are of great interest in the study of the controversial subject of primary meningiomas of the optic nerve sheath.

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Although these arachnoidal clusters are probably the usual sites of origin of meningiomas, it is quite possible that some of these tumors may arise from either the dura or pia-arachnoid, and not only from the arachnoid clusters but from the mesenchymal connective tissue, blood vessels, or melanophores, as well. Globus (1937 and 1944) and also Foot (1940) regarded meningiomas as compound tumors in which either the parenchymatous, epitheloid element or fibroblastic, stromal element may predominate.

Until recently, the study of meningeal tumors was chiefly confined to the benign forms, and the malignant potentiality, while recognized, was thought by most investigators to be rare. In the recent literature greater attention is being focused on the malignant forms. Globus, Levin, and Sheps (1944) and Turner, Craig and Kernohan (1942) found in their respective series that approximately 10 per cent of meningeal tumors were malignant. Malignancy was judged by cell type, tissue architecture, and local invasiveness. Very few instances of metastasis have been reported. There were no cases associated with metastasis in the series of Turner, et al., and only one case of metastasis in the series of Globus, et al.

Two cases are reported of meningioma arising within the intracranial cavity and invading the paranasal sinuses. One of the two cases reaffirms the view of Mallory (1920) that the arachnoid lining cell has fibroblastic potentialities.

CASE REPORTS

CASE 1.—A very large olfactory groove tumor was removed from a white woman, 44 years of age. Preoperatively her vision had been reduced almost to blindness. The roentgenogram showed a marked dipping in the roof of the orbit just anterior to the clinoid process. A right hypophyseal approach was made. A dural growth, much larger than anticipated, was found in the region of the olfactory groove. Lying in the midline, it had pushed far up into the falx. The vascularity of the tumor was pronounced. The posterior border projected into the optic chiasma but there was very little attachment posteriorly. Anteriorly the attachment became increasingly more dense until it became exceedingly dense in the region of the crista galli. "Furious bleeding" from the olfactory groove just to the left of the crista galli was encountered when the tumor was shelled out. The tumor, which weighed 50 grams, was completely removed.

About three and a half years later, a nasal recurrence developed which was treated by diathermy and cauterization. Six years postoperatively, examination showed the left nasal chamber below the lower border of the middle turbinate to be completely filled with a grayish-red polypoid mass. There was complete optic atrophy on the left with moderate proptosis. The roentgenogram showed the left, medial orbital wall to be destroyed, the right ethmoid cells expanded and uniformly dense, and the floor of the frontal sinuses could not be demonstrated. Biopsy specimens were taken from the tumor in the left nasal chamber. A diagnosis of meningioma with extension into left nasal chamber and probable involvement of the left orbit, the left frontal and ethmoid sinuses was made. Surgical removal was recommended but the patient declined operation.

Since that time she has been treated for recurrent severe nasal hemorrhages from the tumor. Two years after the biopsy and eight years after the removal of the intracranial tumor, the growth was found to involve the antrum, ethmoid, nasal cavity, and orbit on the left and was beginning to protrude from the left anterior nares. In spite of this massive involvement the patient's general health apparently continued to be good except for frequent nasal hemorrhages. This follow-up in November, 1944, unfortunately, is the last one available.

Histologic Examination.—The tissue taken from the left nasal chamber has striking characteristics. A predominantly meningothelial-type tumor with well-defined whorls is seen (Fig. 1). There is a covering of well-preserved ciliated respiratory epithelium. The tumor cells are uniform, fusiform, and have oval nuclei with speckled chromatin (Fig. 2). Occasional mitotic figures are seen; there are no giant cells. The fibroblastic, stromal element is limited and tends to lobulate the meningothelial cells (Fig. 3).

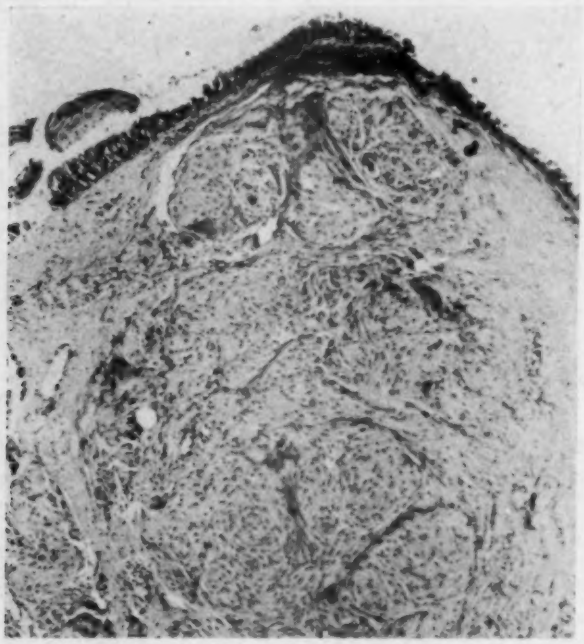


Fig. 1.—Case 1. Section through tumor in nasal chamber showing well-defined whorl formation and covering of respiratory epithelium. (H. & E., ×100, Neg. 94866.)

Examination of the original operative specimen from the brain tumor* shows a similar picture except that whorl formation is not quite as definite (Fig. 4), and there are large areas where the meningothelial cells are arranged in cytoplasmic sheets with ill-defined cell boundaries (Fig. 5). However, here again both architectural pattern and individual cell pattern are uniform.

Morphologically, this tumor is a typical meningothelial meningioma with whorl formation, and histologically, it is benign.

*Examination of this section was made possible through the courtesy of the Department of Pathology, Johns Hopkins University.

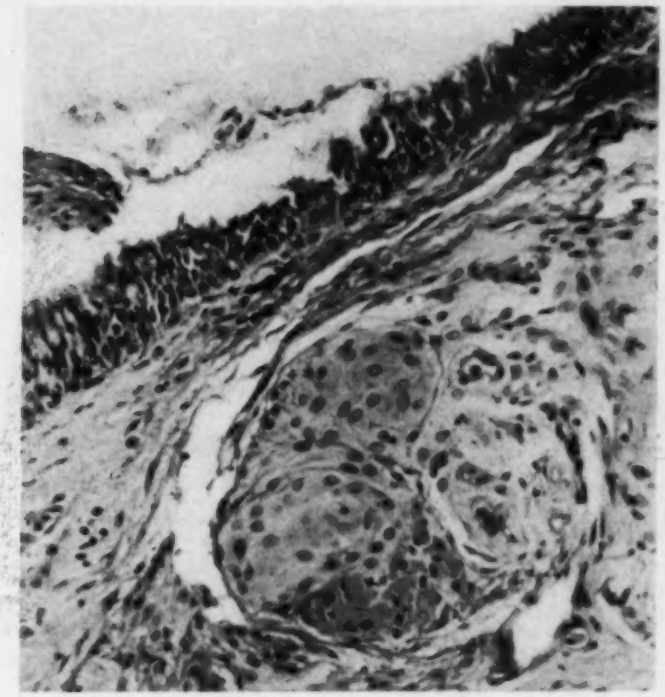


Fig. 2.—Case 1. The covering epithelium is ciliated. The whorls are made up of typical meningothelial cells. (H. & E., ×330, Neg. 94865.)

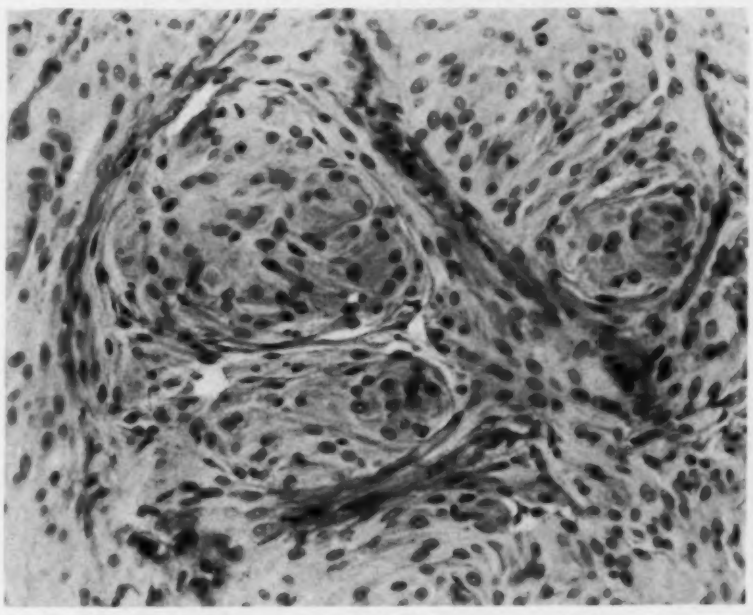


Fig. 3.—Case 1. Lobules made up of one or more whorls are circumscribed by delicate fibrillar bands of collagen. (H. & E., ×330, Neg. 94875.)

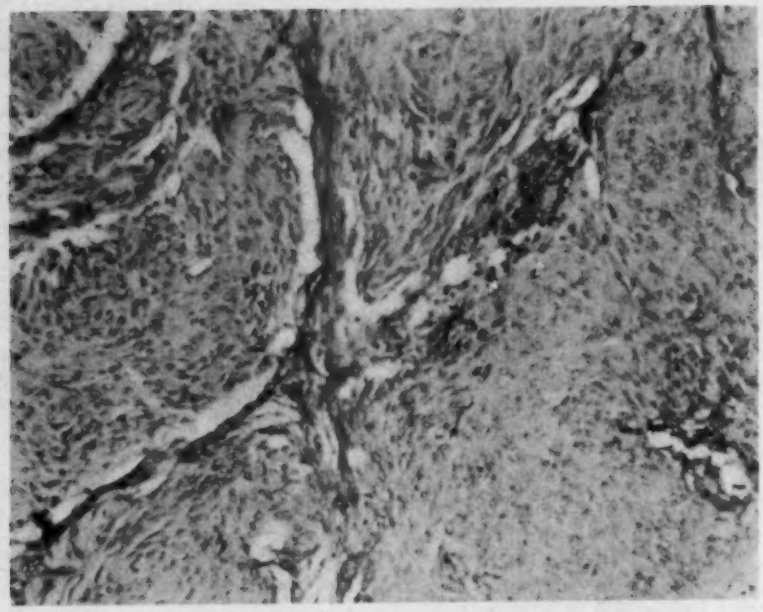


Fig. 4.—Case 1. Section through the intracranial tumor. Cells are similar but whorl formation is not as definite. (H. & E., ×175, Neg. 94864.)

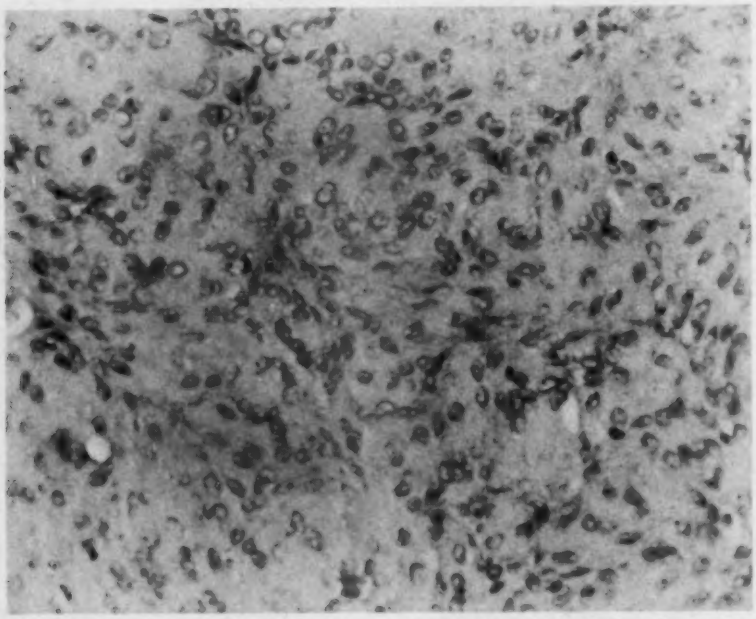


Fig. 5.—Case 1. Note the cytoplasmic sheets with ill-defined cell boundaries. (H. & E., ×435, Neg. 94871.)

CASE 2.*—A Negro man, 58 years of age, was found along the highway, disoriented and incoherent. There was marked facial asymmetry, pronounced exopthalmos on the left, and the left nostril was filled with a hardened tumor.

The roentgenogram was interpreted as follows:† "Left lateral film [Fig. 6] shows clouding in the frontal sinus region, an intense density overlying the ethmoid region and obscuring the sphenoid sinus as well. The sella turcica shows definite erosion especially at the dorsum sellae and posterior clinoid process. Nose-chin, P-A view [Fig. 7] shows an intense density of the left maxillary antrum, the left orbit and the nasal cavity. The medial wall of the orbit is ill-defined and the left frontal sinus shows considerable haziness."



Fig. 6.—Case 2. The sella turcica is eroded and there is cloudiness of the sphenoid and ethmoid sinuses. (Neg. 69740.)

The left eye was enucleated but no intraorbital tumor was found. An eroding neoplasm presented at the medial wall of the orbit and appeared to arise in the ethmoidal labyrinth. Six days later the patient died. At autopsy the left temporal and left frontal poles of the brain were found to be compressed by an adherent encapsulated tumor, measuring 8 by 5 by 2 cm. (Fig. 8). This tumor was densely adherent to the dura in the mid-portion of the left anterior fossa; the brain was not infiltrated but the tumor was somewhat adherent to the pia-arachnoid. The sella turcica was eroded through, and the underlying sphenoid sinus was completely filled with a nonadherent mass. The left ethmoid sinus was likewise filled with new growth. A tumor the size of a hen's egg occupied the

*This case was registered by Dr. W. O. LaMotte, Wilmington, Delaware.

†The x-rays were interpreted by Major C. D. Naidorf of the Army Institute of Pathology staff.



Fig. 7.—Case 2. A nose-chin view shows an intense density of the left orbit, antrum, and nasal cavity. (Neg. 69741.)

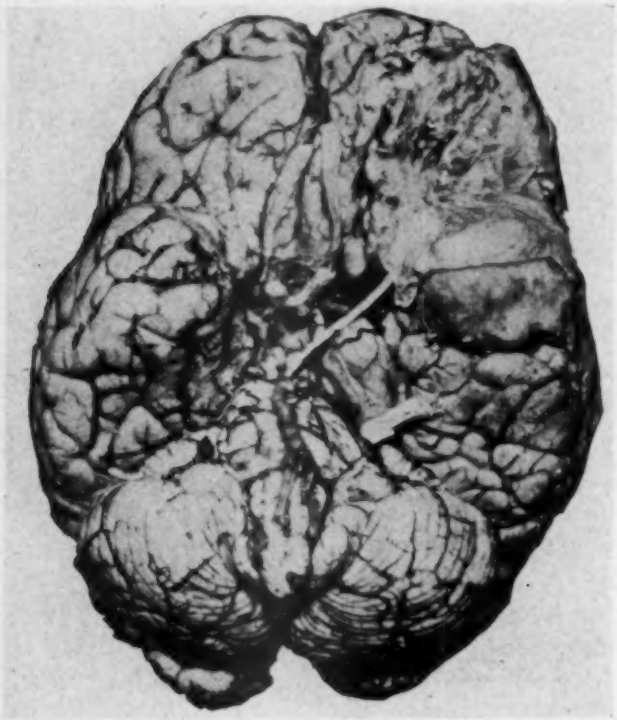


Fig. 8.—Case 2. The meninges are reflected to show an encapsulated tumor compressing the left temporal and left frontal poles of the brain. (Neg. 94863.)

superior portion of a large cavity resulting from operative removal of the nasal septum and "apparent deficiency" in the medial wall of the left antrum. Tumor was also found in the left nasal cavity. On the right side, the sinuses were normal. There was no other evidence of neoplasia at autopsy.

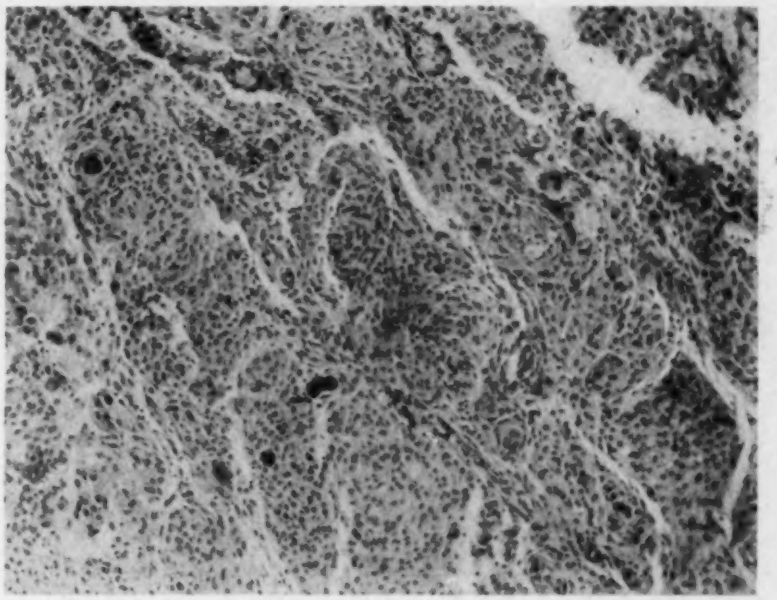


Fig. 9.—Case 2. Section from a portion of the intracranial tumor showing well-developed whorls and psammoma bodies. (H. & E., ×125, Neg. 94869.)

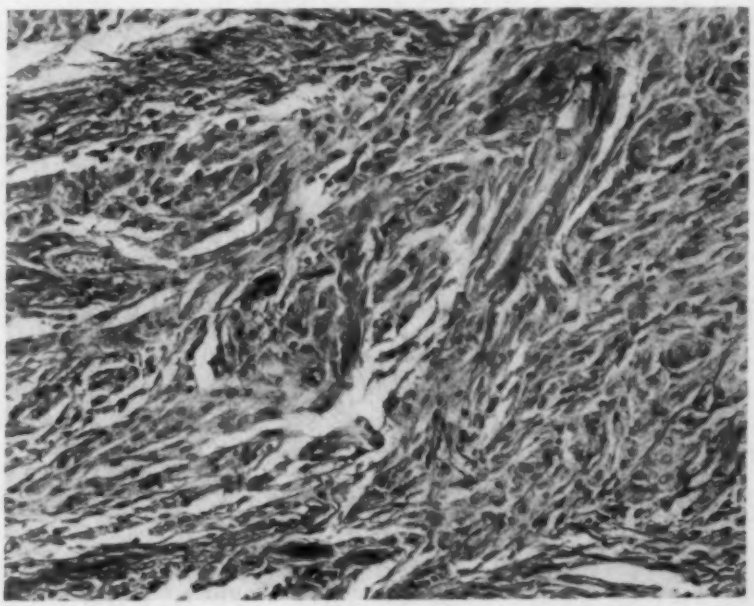


Fig. 10.—Case 2. A transitional area in which there is a loss of architectural pattern, the stromal element becoming more evident. Note the delicate strands of collagen. (P.T.A.H., ×145, Neg. 94991.)

Histologic Examination.—Sections from the intracranial tumor are those of a densely cellular, moderately vascular neoplasm. In general, there is a predominance of a meningothelial-type cell with a slight stromal element. Individual sections examined show a transition from well-developed whorls and psam-

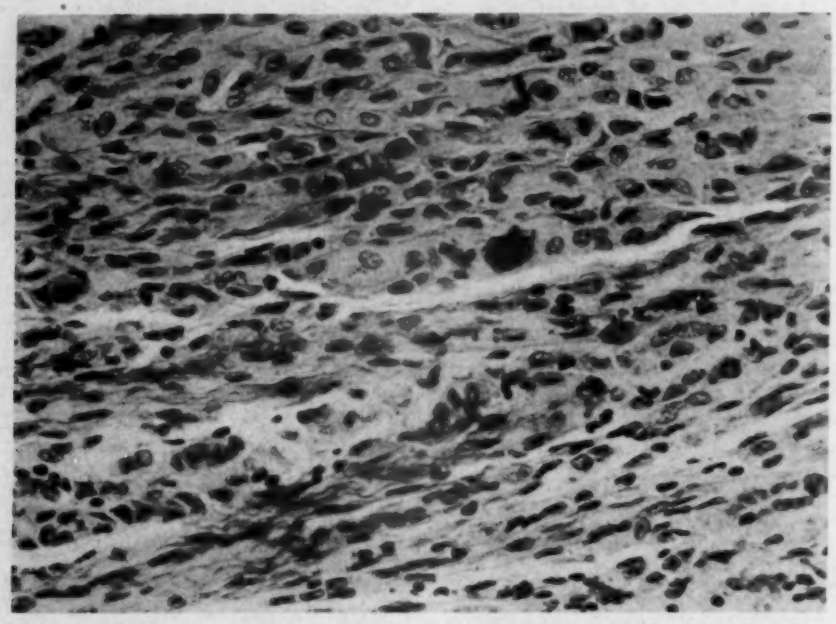


Fig. 11.—Case 2. Transitional area, early giant-cell formation, and slight pleomorphism. Fibrils are recognizable. (H. & E., ×415, Neg. 94867.)

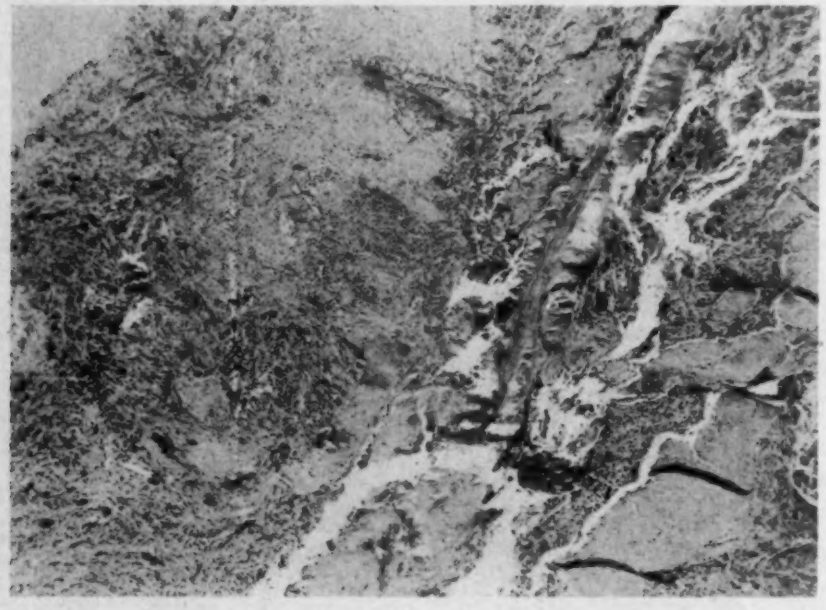


Fig. 12.—Case 2. Appearance of the tumor as it infiltrates the dura. (H. & E., ×60, Neg. 94870.)

moma bodies in some areas (Fig. 9) to other areas where there is a disruption of architectural pattern accompanied by an increase in stromal tissue (Fig. 10). In these latter areas there is slight pleomorphism, hyperchromatism, and occasional early giant-cell formation (Fig. 11). Mitosis is not a feature. The dura is infiltrated (Fig. 12).

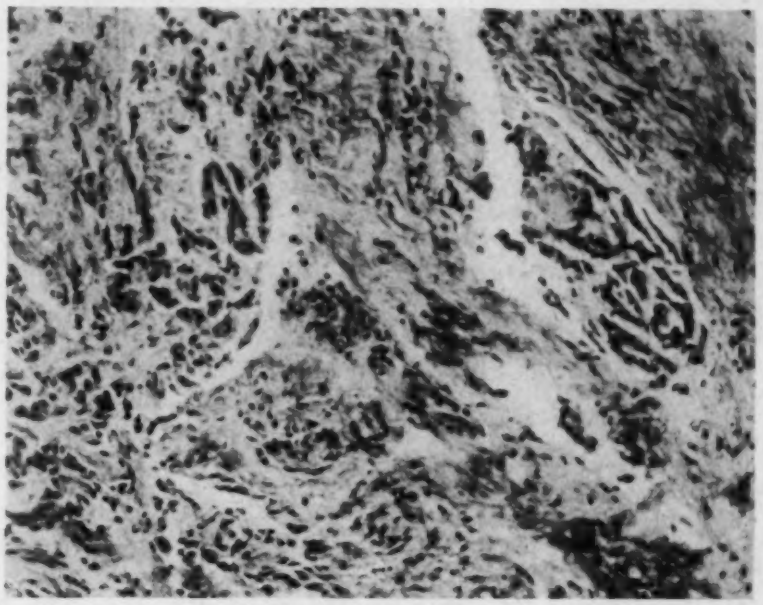


Fig. 13.—Case 2. Section from tumor in nasal cavity. The stromal element predominates and only occasional clusters of meningothelial cells are seen. (H. & E., ×200, Neg. 94872.)

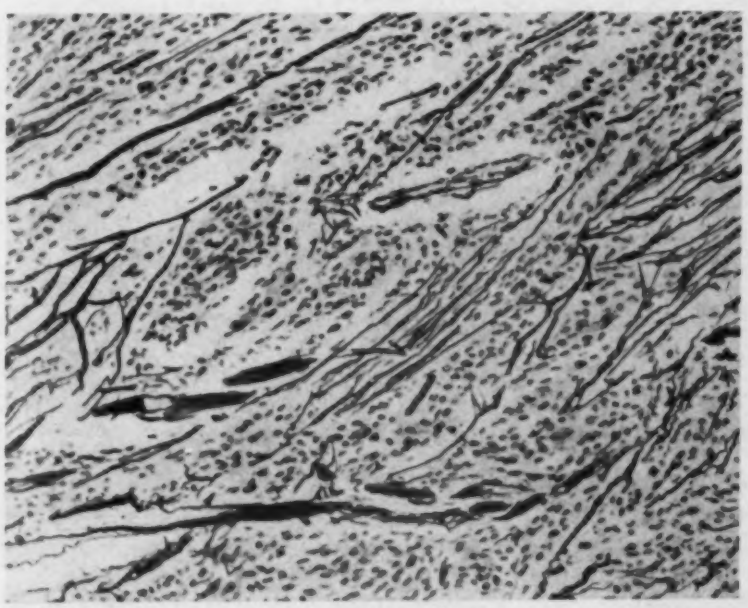


Fig. 14.—Case 2. Reticulin pattern is of the "broken-twig" type and is not a tumor element. (Wilder reticulum, ×165, Neg. 95047.)

Sections taken from the nasal cavity show a striking predominance of stromal element. Here there is a streaming, interlacing pattern of fibroblastic tissue. Meningothelial cells, while present, are very scarce, and whorl formation at the most is abortive (Fig. 13).

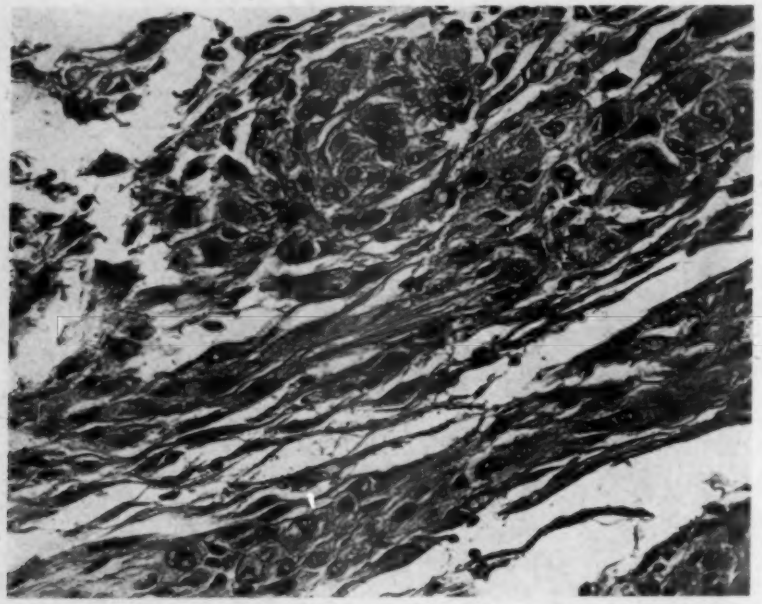


Fig. 15.—Case 2. This section shows the transition between the meningothelial cells making up the whorls and the spindly fibrillar cells of the stroma. (P.T.A.H., ×475, Neg. 95049.)

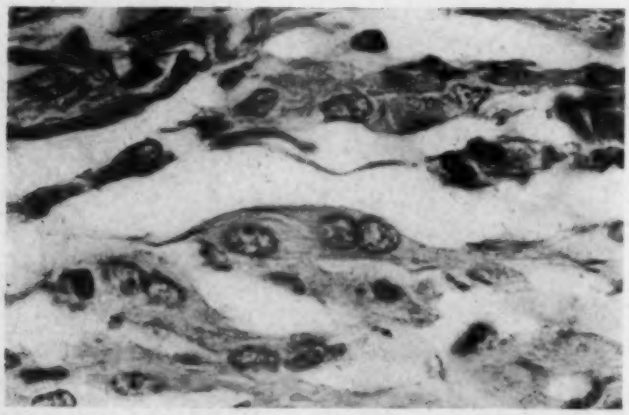


Fig. 16.—Case 2. Fibroglia fibrils issuing from the spindle-shaped meningothelial cells. (P.T.A.H. ×1000, Neg. 95048.)

In the areas in which whorl formation predominates, collagen is concentrated about the vessels; however, a slight amount of collagen can be seen scattered in approximation to the meningothelial cells. In areas where the meningothelial architecture is disrupted, the collagen is increased (Fig. 10) and in sections from the nasal cavity, collagen formation reaches its maximum.

There is very little reticulin formation; coarse fibers of the "broken-twig" type are noted particularly near the area of dural invasion (Fig. 14). These are interpreted as secondary to tumor invasion rather than as a part of the neoplastic growth.

The meningothelial cells making up the whorls contain fibroglia fibrils. A progressive transition can be seen from these polyhedral cells to more spindly cells which are quite similar to fibroblasts and are forming fibroglia fibrils (Figs. 15 and 16).

This tumor is regarded as a meningioma, fibroblastic type. Although it fulfills some of the criteria of Turner, et al. (1942), for low-grade malignancy, it is believed to be histologically benign.

Stains used for sections examined in both of these cases included hematoxylin and eosin, Masson, phosphotungstic acid-hematoxylin, and Wilder reticulum.

DISCUSSION

The exact pathway and mechanism of spread have not been conclusively demonstrated in these two cases. The study of the tumor as it penetrated through bone would have been most interesting, but, unfortunately, no tissue which would clarify this point was available for examination. The records, however, certainly indicate that in each case the meningioma arising primarily in the intracranial cavity infiltrated through the base of the skull to present in the paranasal sinuses and nasal cavity. In Case 1 the bleeding from the olfactory groove at operation, in light of future events, may be interpreted as possibly due to the tumor invading the cribriform plate. The probable pathway in this case was extension through the cribriform plate into the ethmoidal labyrinth. Similarly, in Case 2 the ethmoids were the probable primary pathway of spread, and the eroded sella turcica and invaded underlying sphenoidal sinuses are regarded as a secondary pathway.

Two somewhat similar cases have been reported in the literature. Cushing and Eisenhardt (1938) in their series reported one case in which a right prefrontal meningioma was found at operation to have invaded the frontal sinus, the posterior wall of the sinus having been absorbed. Removal was successful and a twelve-year follow-up found the patient in good health except for impaired sight and occasional convulsive seizures. Sections taken from the sinus showed a covering of ciliated mucous membrane similar to Case 1. Cellular whorls were noted and, in addition, multinuclear masses which resembled giant cells. "The tumor showed no malignant tendencies."

Von Uiberall and Wulfson (1938) described a case in which the presenting symptoms were those of an atypical trigeminal neuralgia. At operation, a "dural endothelioma" was found to have infiltrated into the sphenoidal sinus. Histologically, the tumor was dense and characterized by abundant connective tissue which was infiltrated by "dural endothelioma." Unfortunately, the microscopic description was not given in great detail; however, it would appear that this was a case of meningioma in which the stromal component was predominant.

SUMMARY

Two cases of meningioma are reported which penetrated the base of the skull and invaded the paranasal sinuses. In Case 1 the histologic picture was typical meningothelial meningioma with whorl formation. Case 2 showed a transitional picture of a fibroblastic-type meningioma in which the fibroblastic component became increasingly more evident as the tumor spread to the nasal cavity. In this case of meningioma of the fibroblastic type, there is morphologic evidence that the stromal component was derived from the meningothelial cells which are shown to have the capacity to differentiate into cells that form fibroglia fibrils, lay down collagen, and ultimately become morphologically indistinguishable from fibroblasts. This finding in Case 2 supports the concept that the arachnoid lining cell has fibroblastic potentialities. In Case 1 the biopsy specimen is so fragmentary as to exclude interpretation as to histogenesis. No conclusion can be drawn from this one case as to the frequency with which this potentiality is manifested, but it suggests that the ultimate morphologic appearance of meningioma of the fibroblastic type is the result of this inherent capacity of the arachnoid lining cell.

The tumors in both Case 1 and Case 2 are regarded as benign. Admittedly the behavior of these two tumors was unusual for meningiomas, but the type of spread is regarded as resulting from active tumor growth in a confined space, extending in lines of least resistance, rather than as a manifestation of malignancy.

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EOSINOPHILIC GRANULOMA OF BONE WITH PRIMARY ORAL MANIFESTATIONS

Report of Three Cases

Major Marcus E. Cox, M.C.

IN 1940, Otani and Ehrlich[14] described a lesion which they chose to call solitary granuloma of bone. In that year also, Lichtenstein and Jaffe[12] described the same condition, calling it eosinophilic granuloma of bone. Characterisite was a single, well-localized, sharply defined destructive lesion of granulomatous nature, occurring in either the membranous or the long bones, beginning in the medullary cavity and spreading to the cortex. Histologically, the lesion was characterized by tumorlike aggregates of large macrophages, earmarked by the presence of collections of eosinophilic leucocytes. Otani and Ehrlich[14] suspected that trauma played a role in pathogenesis; Lichtenstein and Jaffe[12] regarded the lesion as probably of infectious origin. Clinically, the onset was abrupt, the course progressive and sometimes complicated by fracture at the site of involvement or extension through the cortex of the involved bone, giving rise to local symptoms, most often pain and tenderness. The disease was regarded as one of childhood, adolescence, and early adult life. At first it was thought that the characteristic lesion was of solitary occurrence, but it soon became apparent that multiple lesions occurred. Green and Farber[8] reported a series of 10 cases, in 4 of which the lesions were solitary and in 6 multiple. Farber[3] declined to accept eosinophilic granuloma as an altogether distinct and separate entity among the granulomatous lesions of bone but regarded it rather as a variant or manifestation of the basic disease process underlying Letterer-Siwe and Hand-Schüller-Christian diseases. In 2 of the 6 cases with multiple bone involvement reported by Green and Farber, lesions of the mandible were observed. The cranial bones, ribs, and vertebrae were the bones which were most often the sites of multiple granulomas. In addition, there has been a considerable amount of literature dealing with the manifestations and underlying conditions of diseases of this nature.[2, 5, 7, 9, 11, 13, 19]

Bailey and Freis[1] have recently reported a case of eosinophilic granuloma with multiple bone involvement in which the lesion in the mandible afforded an opportune site for establishing the diagnosis by biopsy. In a case of eosinophilic granuloma reported by Thoma,[16] the lesion in the mandible preceded recognizable involvement of other bones which followed and which was accompanied by diabetes insipidus. Salman and Darlington[15] reported the case of an 8-year-old boy with an oral lesion in which the clinical symptoms and roent-

genologic evidence suggested Hand-Schüller-Christian disease. Later histologic examination proved the lesion to be eosinophilic granuloma.

Since lesions of the mandible or maxilla may produce the presenting symptoms in either Hand-Schüller-Christian disease or eosinophilic granuloma of bone, it is appropriate to report four cases of eosinophilic granuloma from the Registry of Dental and Oral Pathology of the American Dental Association at the Army Institute of Pathology.

CASE REPORTS

CASE 1 (A. I. P. Acc. No. 163525).—A white soldier, 28 years of age, first noted painless swelling of the left side of the face in September, 1945. Examination revealed a mass in the left side of the body of the mandible, just anterior to the ramus. Roentgenologic examination gave the impression of possible osteogenic sarcoma.

Two years prior to the appearance of the tumor of the mandible, the patient had received a light blow on the jaw. Two days later a dentist had removed a tooth which, the patient stated, had been loose for some time, making extraction very "easy." The remainder of the past medical history was irrelevant.



Fig. 1.—Case 1. Roentgenogram showing appearance of lesion of mandible. (A.I.P. Neg. 95031.)

Roentgenologic examination, Oct. 25, 1945, at another hospital showed that the area of bone destruction in the left side of the body of the mandible had questionable invasive properties (Figs. 1 and 2). The cystlike lesion involved the roots of the last molar tooth on the left and had almost completely destroyed

the inferior and lateral borders of the mandible. There was no evidence of new bone formation. No variation in density of the overlying soft tissues was noted. Roentgenograms of the chest revealed a punched-out area of rarefaction in the posterior segment of the tenth rib on the left. This was comparable in general appearance to the lesion of the mandible except for rather definite evidence of new bone formation. The remainder of the physical examination was negative.

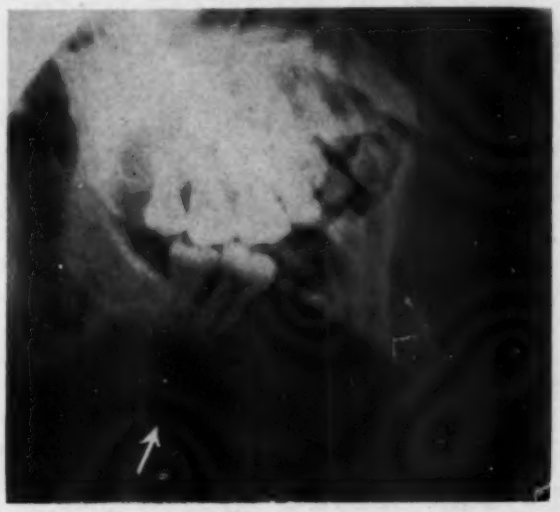


Fig. 2.—Case 1. Roentgenogram showing extent of destruction of inferior border of mandible. A.I.P. Neg. 95028.)

Routine laboratory examinations showed that leucocytes ranged from 11,800 to 14,200, with polymorphonuclear neutrophiles ranging from 45 to 20 per cent, lymphocytes from 32 to 51 per cent, and eosinophiles 4 to 8 per cent. Urinalyses were negative except one specimen in which numerous white blood cells were present.

On Nov. 6, 1945, curettement of the cystic lesions of the mandible yielded soft yellowish tissue and purulent material. Several small sequestra were removed and a bone graft from the ilium was laid in the defect. In a period of eleven days in November, 1945, the patient was given four x-ray treatments over the mandible with a total dosage of 1200 r. The postoperative course was uneventful.

Pathologic Examination.—The material was yellowish-brown, partly necrotic, and had the general appearance of inflammatory tissue. Microscopically, some fragments were seen to be comprised almost entirely of closely packed aggregates of eosinophiles intermingled with sheets of large, round, irregularly shaped, polygonal cells with homogeneous nuclei and pale slightly foamy cytoplasm (Fig. 3). Occasional multinucleated giant cells were encountered. There was considerable vascularity and many areas of hemorrhage. Other fragments were composed of a dense fibrous tissue in which the mononuclear cells and eosinophiles were less conspicuous.

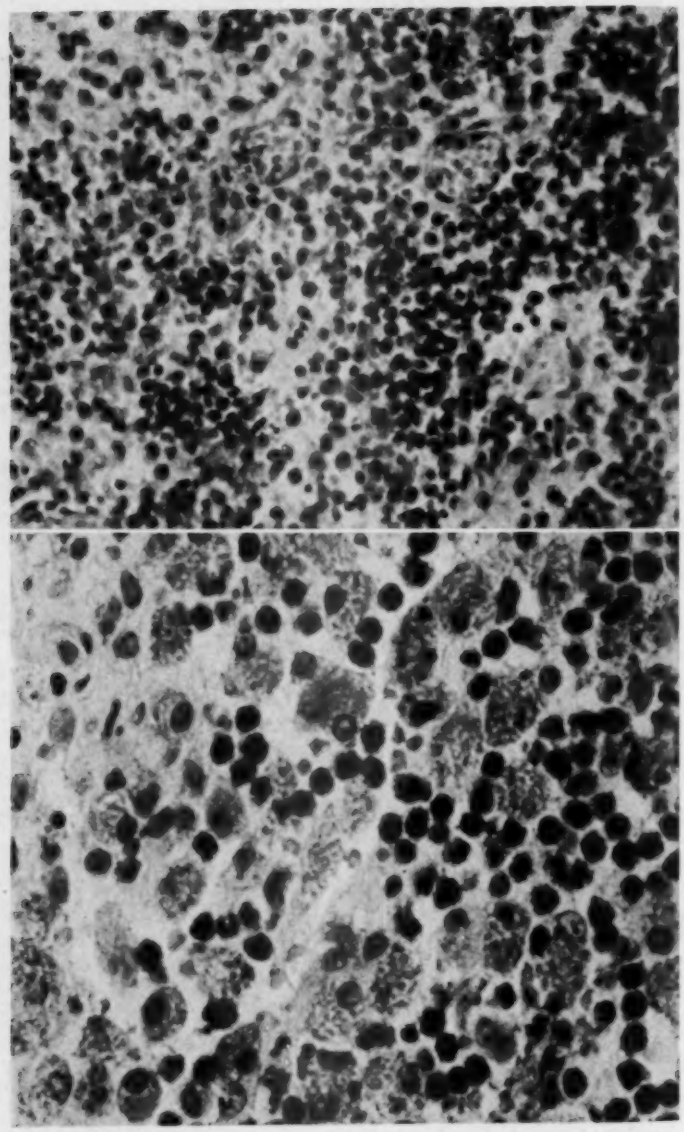


Fig. 3.—Case 1. *A*, Loose collections of eosinophiles dominate the microscopic picture in some fields. (A.I.P. Neg. 95180. Hematoxylin and eosin stain. ×350.) *B*, High-power view showing the relative proportions of macrophages and eosinophiles. (A.I.P. Neg. 95302. Hematoxylin and eosin stain. ×705.)

CASE 2 (A.I.P. Acc. No. 147994).—A white soldier, 28 years of age, was first seen in the dental clinic in April, 1944, because of soreness of the gums of the maxillary arch. Roentgenograms revealed apical abscesses of the right upper premolars. Following extraction of the teeth the soldier was returned to duty.

Subsequently, surface ulceration developed about the sites of extraction, progressed, and ultimately exposed the entire length of the right upper first and second molars. Roentgenologic examination disclosed bone resorption of the alveolar processes of this area, cloudiness of the left antrum and ethmoid cells, but no lesions of the long bones or the thoracic cage. Because the oral condition did not improve on local treatment, a course of penicillin was given in October, 1944, with resultant clinical benefit. Later, the upper lateral incisor, second and third molars, and the lower second and third molars on the right, and the upper third molar, lower second premolar, and second and third molars on the left were removed.

Repeated bacteriologic studies of the gum lesions sometimes showed beta hemolytic streptococci and fusiform bacilli. Monilia was obtained in culture from the gums on one occasion.

Two previous biopsies of the gums, Sept. 4 and Oct. 5, 1944, were interpreted as chronic inflammatory tissue devoid of histopathologic specificity.

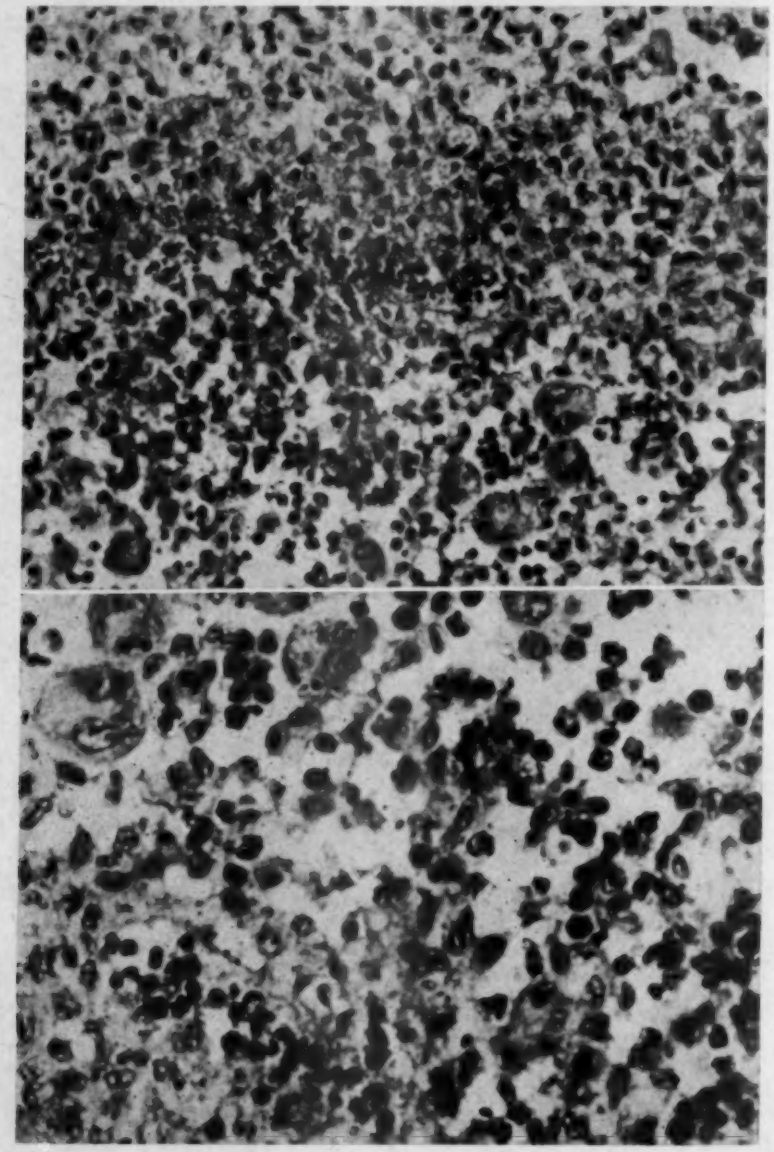


Fig. 4.—Case 2. Roentgenogram showing character and extent of the involvement of the mandible. (A.I.P. Neg. 95035.)

On Nov. 27, 1944, the patient was transferred to another hospital center for thorough study. Slight palpable enlargement of the anterior cervical lymph nodes comprised the only additional physical finding. Further roentgenologic study showed multiple radiolucent areas of bone destruction of the mandible about the sites of tooth extraction without evidence of new bone forma-

tion. The rami of the mandible were porotic. Roentgenologic survey of the skeleton revealed a fusiform swelling of the lower third of the right tibia.

Extension of the oral ulceration required the extraction of several more teeth. The administration of sulfonamides, penicillin, mapharsen and local applications were without evident beneficial effect. A biopsy of the gums, performed Jan. 6, 1945, was reported as inflammatory tissue, another on March 3, as acute and chronic osteomyelitis. It is significant that it was noted that the inflammatory tissue was heavily laden with eosinophiles.



A.

B.

Fig. 5.—Case 2. *A*, Low-power view showing prominence of eosinophiles and the presence of multinucleated giant cells. (A.I.P. Neg. 95179. Hematoxylin and eosin stain. ×350.)

B, High-power view showing the character of the giant cell forms. (A.I.P. Neg. 95301. Hematoxylin and eosin stain. ×705.)

Review of roentgenograms and re-examination on Sept. 10, 1945, disclosed radiolucent areas of bone destruction of the alveolar ridge between the right lower cuspid and second molar, left lower first premolar and third molar without lateral expansion of the mandible, and destructive lesions of the rami and symphysis without surrounding bone reaction (Fig. 4). There was destruction of the right maxilla and lateral wall of the antrum. A large defect of the base of the skull involved the body of the sphenoid, the dorsum sellae, and both petrous pyramids. In addition, there was an area of destruction of the second cervical vertebra with apparent extension into the odontoid process and a circumscribed defect in the right wing of the sacrum which was irregular in outline and without evidence of surrounding bone reaction. The widening of the shaft of the lower third of the right tibia persisted.

Routine laboratory examinations revealed no findings of note.

Between Oct. 1 and 12, 1945, the patient was given irradiation therapy as follows: 500 r. to the right lateral skull, 500 r. to the left lateral skull, 600 r. to the right mandible, 300 r. to the left mandible, 400 r. to the right maxilla, 600 r. to the left sacrum.

Pathologic Examination.—The gross specimen consisted of fragments of firm gray tissue covered with mucous membrane removed from the region of the lower right second molar. On microscopic examination the surface epithelium was hyperplastic and the subjacent stroma was infiltrated by an acute and chronic inflammatory cellular exudate. In the deeper portions of the section a rather diffuse histiocytic reaction was overshadowed by aggregates of eosinophiles. The cytoplasm of the histiocytes was rather foamy in some fields (Fig. 5).

Case 3 (A.I.P. Acc. No. 133904).—A white soldier, 20 years of age, with two years of military service, was transferred to a maxillofacial center after routine dental examination in the field disclosed a large cystic defect in the symphysis of the mandible. About six months prior to induction the patient had had several upper and lower teeth extracted because they had become loose and painful. About six months later several anterior and most of the posterior teeth were extracted. Cystic cavities in the lower cuspid areas on either side interfered with the wearing of dentures. The past medical history was otherwise irrelevant.

Physical examination disclosed a large cavity in the right side of the body of the mandible which opened onto the ridge in the area of the lower right central incisor. The cavity was filled with food debris and appeared to be chronically inflamed. All routine laboratory tests yielded findings within normal limits.

On Jan. 3, 1945, the cystic lesion of the mandible was curetted and found to be more extensive than anticipated. The external plate was missing from the lower right cuspid to the lower left second premolar. The material removed was rather friable, and had the appearance of granulation tissue.

The result of a roentgenologic survey of the entire skeleton was as follows: a number of sharply marginated osteolytic defects throughout both right and

left sides of the body of the mandible, two irregular lytic defects in the left parietal bone, six similar areas in the frontal bone; an elongated irregularly shaped cystic defect in the mid-portion of the right clavicle, extensive coalescent defects in the lower halves of the femurs, and one small cavity in the shaft of the right fibula. There was no apparent reaction of the bone surrounding any of these lesions.

The results of blood chemistry studies were as follows: calcium was 10 mg. per cent; phosphorus, 4.8 mg. per cent; phosphatase 2.2 Bodansky units (first examination), 7.2 Bodansky units (second determination); total protein, 6.5 Gm. per cent; serum albumin, 3.8 Gm. per cent; globulin 2.7 Gm. per cent; A/G ratio, 1.4/1; alkaline phosphatase, 5.7 Bodansky units; acid phosphatase, 2.7 Bodansky units; cholesterol, 185 mg. per cent. The sedimentation rate ranged from 18 to 30 mm. per hour. The results of all other laboratory tests were within normal limits.

Pathologic Examination.—The curettings from the first biopsy of the mandibular lesion consisted of several small portions of reddish-brown tissue. Sections showed a highly cellular matrix composed of mononuclear cells with round, somewhat indented nuclei. At one margin of the section several multinucleated giant cells were noted. Within the cytoplasm of these cells was considerable phagocytosed eosinophilic material and yellow pigment. The presence of large numbers of eosinophiles was a conspicuous feature.

The second biopsy from the mandible showed a similar picture. Some areas seemed to be comprised of almost solid sheets of eosinophiles. Giant cells were not as conspicuous as in the original specimen.

Microscopically, the appearance of the lesion of the right clavicle was similar to that of the jaw. Several clear spaces of considerable size apparently contained fluid; the stromal cells were relatively uniform. Infiltration with eosinophilic leucocytes was again a prominent feature.

Smears of sternal marrow were normal.

COMMENT

Each of these patients sought medical attention as the result of symptoms stemming from lesions in the anatomic realm of the oral surgeon. Their cases emphasize the fact that eosinophilic granuloma must be differentiated from other lesions of bone, both inflammatory and neoplastic. In Case 1, the impression at the time of the first investigation was that the lesion was osteogenic sarcoma. In Case 2, inflammatory entities, such as tuberculosis and blastomycosis, were suspected after study revealed multiple widely disseminated bone defects. Furthermore, these three cases serve to emphasize that multiple bone involvement may be missed unless the nature of the lesion giving rise to the presenting symptoms is recognized and a roentgenologic survey of the skeletal system made. It seems probable that in Case 2 at least, some of the extraoral lesions developed during the period when those of the maxilla and mandible were under treatment. The fact that the diagnosis in each case was established by biopsy serves to stress the importance of submitting all tissue removed from oral lesions for

histologic examination. Other laboratory examinations were largely noncontributory. Only in the first case was there mild eosinophilia of the peripheral blood, a feature which is supposed to be rather common in cases of eosinophilic granuloma of bone.

The results of irradiation therapy in the last two cases are as yet unknown. The patient in Case 1, with only two bones involved, apparently made an uneventful recovery following irradiation. According to Farber[4] it is becoming increasingly apparent that some cases do not respond to irradiation as readily as might be expected from some accounts in the literature. Accordingly, the prognosis in eosinophilic granuloma of bone should be more guarded.

Attempts to isolate a likely pathogen from the cases presented in this report proved indecisive. There was no histopathologic evidence to indicate, or to suggest, a nonbacterial pathogen.

The cases presented are not intended to furnish the basis for a definitive study of eosinophilic granuloma of bone as an entity, or its status as a variant of an underlying disease process which may be common to Hand-Schüller-Christian disease and other forms of idiopathic xanthomatosis. However, the clinical manifestations and histologic pictures were consistent with what are regarded as characteristic of eosinophilic granuloma. Gross and Jacox[9] made note of the frequency with which eosinophilia is encountered in Hand-Schüller-Christian disease. It is notable that the case of Salman and Darlington[15] presented the triad of Hand-Schüller-Christian disease but the biopsy was regarded as typical of eosinophilic granuloma. It is only through follow-up studies of cases such as these, in which there is apparent overlapping of the two conditions, that a clear understanding of the underlying processes may be achieved.

If one regards transformation of macrophages into lipophages, resulting in a frankly lipogranulomatous type of lesion, as the histopathologic hallmark of Hand-Schüller-Christian disease, then the three cases presented may be considered as examples of eosinophilic granuloma by virtue of its absence. Most proponents of the belief that eosinophilic granuloma and Hand-Schüller-Christian disease are merely two stages of the same process feel that the lesion of eosinophilic granuloma assumes a lipomatous character later in the course of the disease. The belief expressed by Jaffe and Lichtenstein[10] that the lesions of eosinophilic granuloma may regress and heal spontaneously without first passing through the lipogranulomatous stage is interesting. In material secured from oral lesions this feature may be difficult to evaluate, as was evidenced in the biopsy material from this group of cases. The fibroblastic activity noted about the periphery of oral lesions may be the proliferation of repair or may be a proliferative inflammatory response to secondary infection.

Follow-up studies on cases such as these should serve to clarify current notions about the nature of eosinophilic granuloma of bone and of the disease process which seems to underly it and kindred conditions.

SUMMARY

Three cases of eosinophilic granuloma of bone in young adults are reported in which the oral lesions gave rise to the primary symptoms. The occurrence

of lesions in the mandible and maxilla in this disease would seem to strengthen the growing body of opinion that eosinophilic granuloma of bone is a variant of the disease process which underlies Letterer-Siwe and Hand-Schüller-Christian diseases. The importance of biopsy study in cases of this kind is stressed primarily as a diagnostic procedure, and secondily, as a means of obtaining information that will clarify our views on this interesting group of disease variants. The importance of close follow-up study is stressed.

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FIBROUS DYSPLASIA (OSSIFYING FIBROMA) OF THE MAXILLA AND MANDIBLE

MAJOR HANS G. SCHLUMBERGER, M.C.*

INTRODUCTION

A SYNDROME characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females was described in 1937 by Albright and his co-workers.[1] The following year Lichtenstein[6] reported eight additional cases, and on the basis of the bone lesions suggested the term "fibrous dysplasia of bone" to identifying the disease. In 1942 Lichtenstein and Jaffe[7] described an additional fifteen cases and reviewed the literature.

The bone changes are characterized by the replacement of cancellous bone and marrow by a white, firm, somewhat gritty, fibrous tissue. Microscopically the connective tissue may vary considerably in character, but usually it is fairly dense and arranged in interlacing bundles and whorls. Within this fibrous tissue are numerous spicules of bone or osteoid, formed by direct metaplasia of the connective tissue cells. Cysts are uncommon and, when present, are quite small. Unlike the constituents of the blood in von Recklinghausen's or Paget's diseases of bone, the serum phosphorus, calcium, and phosphatase in fibrous dysplasia are within normal limits.

Due primarily to the work of Lichtenstein and Jaffe, fibrous dysplasia is now recognized as a distinct lesion of bone. In the majority of cases it is not associated with the pigmentation of the skin and precocious puberty with which it is combined to make up what is frequently referred to as "Albright's syndrome." Furthermore, with increasing frequency, the lesion is reported as occurring in a single bone.[11] These observations raise the question of whether the lesions of the maxilla and mandible identified as ossifying fibroma are also examples of fibrous dysplasia.

For a long time the localized connective tissue overgrowth in the jaws, known as ossifying fibroma, was grouped with osteitis fibrosa localisata or even with the generalized form of osteitis fibrosa described by von Recklinghausen. In 1931 Becks and Weber[2] observed similar changes in the alveolar bones of dogs kept on a diet low in calcium and vitamin D. They denied the possibility that the changes were a form of rickets and identified them as "osteodystrophia fibrosa (intra-osseous epulis)." Geschickter and Copeland[3] regarded ossifying fibroma as true tumors, the majority of which arose in the subperiosteum and only secondarily assumed a central position in the mandible or maxilla. This view has recently received the support of Herzog.[5] Phemister and Grimson[9]

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TABLE I. FIBROUS DYSPLASIA OF THE MAXILLA AND MANDIBLE

CASE NO.	A.I.P.* ACC. NO.	SITE OF LESION	AGE†	HISTORY OF TRAUMA	DURATION OF SYMPTOMS	LOCAL SWELLING	LOCAL TENDERNESS	X-RAY DIAGNOSIS	PATHOLOGIC DIAGNOSIS	REMARKS
1	94650	Left maxilla	23	–	4 mo.	+	–		Ossifying fibroma	
2	116477	Left maxilla	27	–	4 mo.	+	–		Periosteal fibro-sarcoma	
3	164352	Left maxilla	22	–	10 yr.	+	–		Reactive bone proliferation	
4	94803	Right maxilla	15	–	9 mo.	+	–		Osteofibroma	Onset of symptoms followed a toothache
5	102554	Right maxilla	26	–	2 mo.	+		Ossifying fibroma	Ossifying fibroma	
6	110804	Right maxilla	24	–	6 mo.	+	–	Fibro-osteoma	Fibro-osteoma	Recent onset of diplopia
7	135364	Right maxilla	34	–	3½ yr.	+	–	Neoplasm	Ossifying fibroma	Growth recurred after initial removal
8	108696	Right mandible	22	–	12 yr.	+	–	Cyst	Fibrous dysplasia	
9	126502	Right mandible	37	–	6 mo.	+	–	Adamantinoma	Ossifying fibroma	
10	91556	Right mandible	22		7 yr. (?)	+	+	Tumor	Osteofibroma	Onset of symptoms with toothache
11	137720	Right mandible	31		1½ yr.	+	+	Cyst	Fibro-osteoma	
12	128472	etc. Right mandible,	33	–	–	–	–	Osteitis fibrosa cystica	Osteitis fibrosa cystica	Case of polyostotic fibrous dysplasia in which mandibular lesion was an incidental finding

*Army Institute of Pathology.

†The sex is male, the race white, except Case 8, a Negro.

also accepted the neoplastic character of the lesion and contended that it bore the same relation to the membranous bones of the jaws as do the chondromas and exostoses to bone preformed in cartilage. In further support of this view they pointed out that fractures of the maxilla or mandible healed by the overgrowth of connective tissue which subsequently underwent direct osseous metaplasia. Hara,[4] in a recent report of three cases occurring in the maxilla, also recognized the lesion as a true neoplasm.

In his original article on fibrous dysplasia, Lichtenstein[6] cited an example of so-called central osteoma of the maxilla which he regarded as histologically identical with fibrous dysplasia. He stated further that several of the cases which Phemister and Grimson[9] reported as fibrous osteoma were indistinguishable from fibrous dysplasia. In 1942 Mallory[8] likewise accepted the probability that ossifying fibromas and fibrous dysplasia are one and the same. This interpretation has also received the support of roentgenologists. In a personal communication, Dr. John D. Camp of the section on roentgenology at the Mayo Clinic wrote that he still calls the lesion fibrous osteoma, but with the qualifying classification of monostotic fibrous dysplasia, since he believes they represent the same condition.



Fig. 1.—Case 5. Fibrous dysplasia of the right maxilla showing diffuse increased density of the bone and right antrum.

MATERIAL

During the war, the Army Institute of Pathology received protocols and tissues from twelve cases of so-called ossifying fibroma of the maxilla or mandible

(Table I). Since in all but one case the patients were members of the Army, the age (average 26 years) and sex (male) is of no statistical significance. Several of the seven lesions that arose in the maxilla encroached on the antrum (Fig. 1). The remaining five involved the body of the right mandible; in one of these, an example of polyostotic fibrous dysplasia (Case 12), similar lesions were found in the ribs, first lumbar vertebra, and sacrum. The absence of involvement of the left half of the mandible may be due to chance, since the series is too small to permit of further interpretation.

REPRESENTATIVE CASES

Case 4.—The patient, a 15-year-old white boy, was a civilian dependent, and the only member of this series not in active military service. After a toothache he noticed that the periorbital tissue of the right eye was swollen. Three weeks later his nasal passages were obstructed, but he suffered no pain or other discomfort. Obout six months after the onset of symptoms the right eye became more prominent; examination revealed that the pupil reacted slowly to light and accommodation. The right nasal passage was obstructed by red edematous tissue which projected from the base of the lateral wall. The septum deviated to the left. In the right orbit was a fixed and indurated palpable mass; the eye was proptosed and displaced laterally and downward. A roentgenogram revealed a diffuse density involving the medial third of the right orbit, right sphenoid and ethmoid sinuses, and the greater part of the right side of the nasal cavity. Biopsy specimens taken from different regions were similar in histologic pattern, which consisted of interlacing bundles of fibrous tissue with varying amounts of osteoid and metaplastic bone.

Case 6.—A 24-year-old white soldier first noticed slight asymmetry of his face six months before hospitalization; three months later a dentist discovered an enlargement of the maxilla above the right premolar. Subjectively the patient was aware of altered tactile sense in the infraorbital area and of impaired vision in the right eye associated with diplopia. The roentgenogram revealed a diffuse increase in density of the right maxilla. Biopsy specimens taken from widely separated regions of the lesion showed an identical histologic structure: a rather cellular connective tissue in which were numerous spicules of metaplastic bone.

Case 10.—Seven years earlier, a 22-year-old white soldier had experienced severe pain in one of the teeth of his right lower jaw. Extraction of the tooth was difficult, and it broke during the operation. About a year later the patient noticed a small mass on the right side of his mandible; the growth slowly increased in size. A recent roentgenogram revealed a large radiolucent area in the right mandible (Fig. 2) which extended from the second molar to and including the first premolar. The lesion was curetted and a firm, white, gritty tissue removed. On microscopic examination this was found to be composed of rather loose connective tissue through which islets of metaplastic bone were scattered.

Case 12.—This is the only example in the present series in which a lesion of the maxilla or mandible was associated with similar changes in other bones.



Fig. 2.—Case 10. Large radiolucent defect in the right mandible, extending from the second molar to and including the first premolar. Histologically the lesion was identified as fibrous dysplasia.



Fig. 3.—Case 12. Diffusely enlarged and irregularly decalcified right mandible in a case of polyostotic fibrous dysplasia.

Unfortunately, a biopsy specimen of the mandibular lesion was not obtained. For seven years a 33-year-old white male had suffered increasing pain in the scapular region. In the roentgenogram a cystlike defect was found in the right scapula; a radiolucent area covered by a thin and expanded cortex was present in the sixth, seventh, and tenth ribs on the right and in the eighth and eleventh ribs on the left. A large irregular region of bone destruction was found in the first lumbar vertebra; three small cysts were seen in the right wing of the sacrum. The right half of the mandible was enlarged and contained several areas of decreased density (Fig. 3) similar to those in the ribs. The serum calcium, phosphorus, and phosphatase were within normal limits. The involved portion of the eleventh left rib was excised; and when it was sectioned, the normal marrow and cancellous bone were found to be replaced by firm gray-white tissue, histologically similar to the bone in fibrous dysplasia.

TREATMENT

Curettement or attempted excision of the lesion was the only therapy employed in these cases. The incidence of recurrence cannot be determined, for in most instances only a few months have elapsed since the operation was performed. Geschickter and Copeland[3] recommended careful excision with cauterization of the surrounding region, during a period of quiescence. Phemister and Grimson[9] believe that in view of the benign nature of the lesion, massive resection, with its accompanying disfigurement and high mortality, is unjustified. They suggest that the tumors be operatively removed as thoroughly as possible without too great destruction of the jawbone. These authors also believe that roentgen therapy serves to inhibit further growth of portions of the lesion not removed at operation. During the discussion of a recent paper by Pugh[10] on fibrous dysplasia of the skull, Sosman stated that the growth of the abnormal tissue could apparently be stopped by roentgen treatment. He recommended the use of 750 to 800 r. but cautioned against excessive doses which might produce brain damage.

DISCUSSION

The answer to the question raised in the introduction of the possible identity of fibrous dysplasia and ossifying fibroma must be sought in the histologic appearance of the two lesions. In some instances the resemblance is close (Fig. 4). Partially calcified trabeculae of bone arise by direct metaplasia within a connective tissue stroma and are indistinguishable in their structure and arrangement from fibrous dysplasia occurring in other bones, e.g., the ribs. The arrangement of the new bone in the form of spherules and the presence of non-ossified deposits of calcium are frequent findings in the so-called ossifying fibromas (Fig. 5). A recent survey of fifty-eight cases of monostotic fibrous dysplasia,[11] exclusive of those in the maxilla and mandible, showed that such spherules of bone and deposits of calcium are also found in lesions of the ribs and long bones where the diagnosis of fibrous dysplasia is not in doubt. An ap-

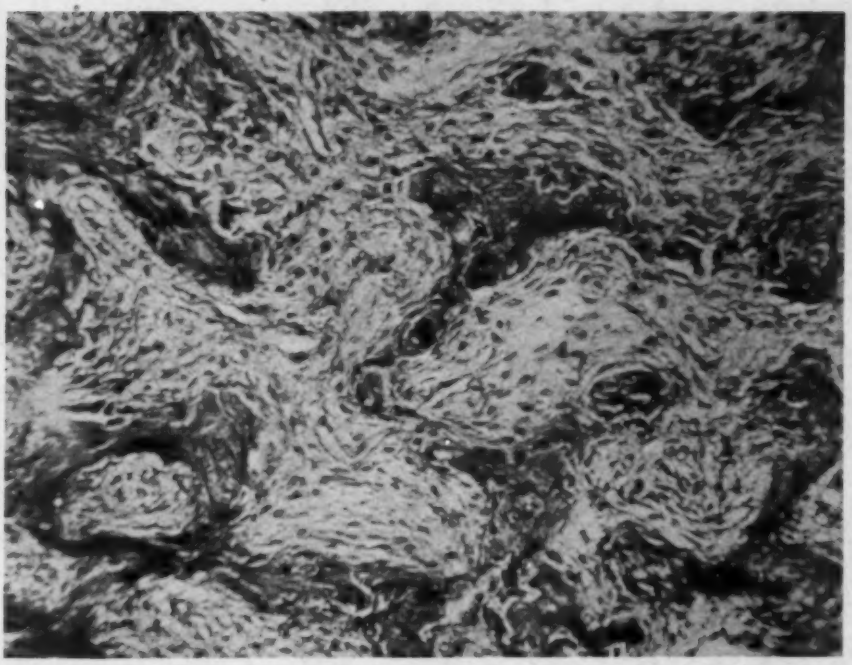


Fig. 4.—Case 1. Metaplastic fibrous bone formation from an area of fibrous dysplasia in the left maxilla. (Masson's trichrome stain. ×230.)

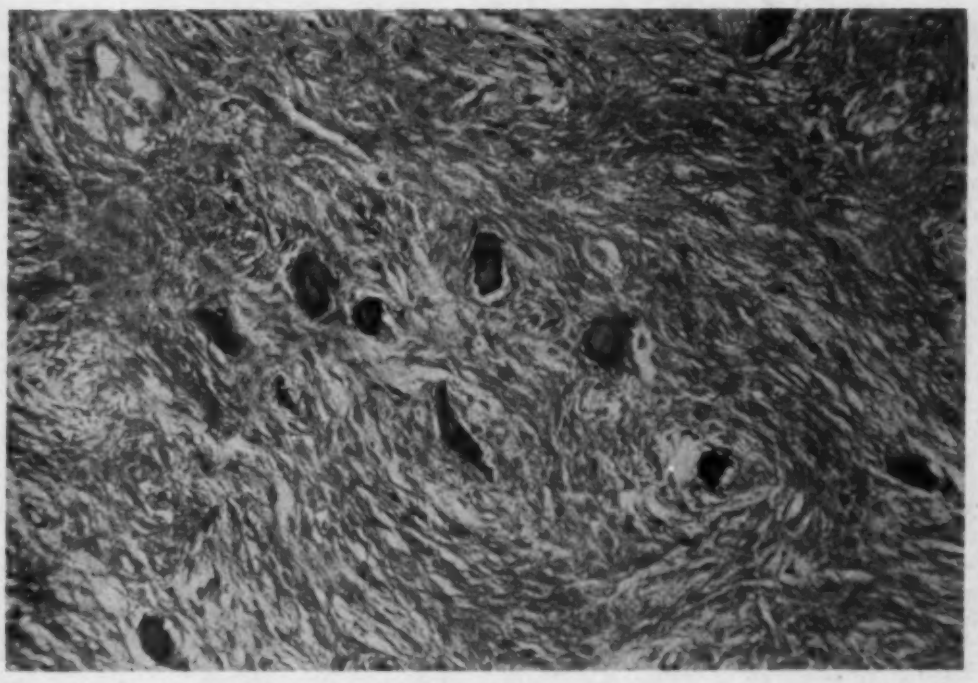


Fig. 5.—Case 7. Fibrous dysplasia of the right maxilla. The islets of metaplastic bone are frequently small and spherical when found in this lesion in the bones of the jaw. (Hematoxylin and eosin stain. ×230.)

pearance such as that presented by Case 4 (Fig. 6) offers features common to both forms of connective tissue ossification. Since, therefore, histologic criteria do not exist by which fibrous dysplasia of the maxilla or mandible can be distinguished from so-called ossifying fibroma of these bones, and because their clinical behavior is identical, it is concluded that the ossifying fibroma is a variant of monostotic fibrous dysplasia.

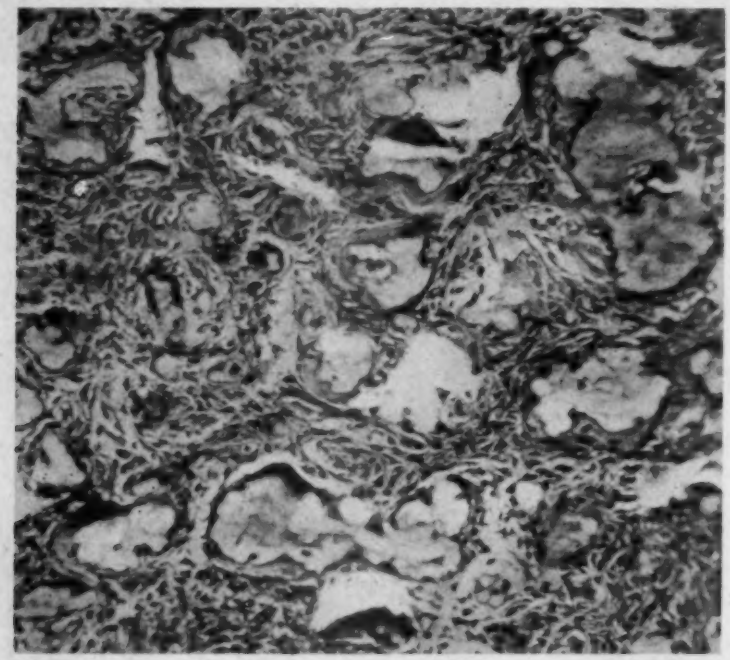


Fig. 6.—Case 4. Irregular deposits of calcium and spicules of metaplastic bone in a lesion of the right maxilla. Histologically this occupies an intermediate position between the cases illustrated in Figs. 4 and 5. (Hematoxylin and eosin stain. ×230.)

SUMMARY

1. Fibrous dysplasia involved the maxilla seven times and the mandible four times in the eleven cases of this series in which the bone lesion was solitary. In the single polyostotic case the mandible was the only bone of the skull affected.

2. On clinicopathologic grounds the so-called ossifying fibroma of the maxilla or mandible may be regarded as a variant of fibrous dysplasia.

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INTRACRANIAL EPIDERMOIDS (CHOLESTEATOMAS)

CAPTAIN WARREN A. BENNETT, M.C., A.U.S.

SINCE the original description of a brain tumor containing hair was given by Verattus in 1745, numerous names have been attached to intracranial epidermoids. Pinson, in 1807, constructed a wax model of an epidermoid tumor in the fourth ventricle and cerebellum which was demonstrated by Dumeril before the Société de Médicine. The confusion and controversy over the pathogenesis began in 1829 when Cruveilheir first described an intercerebral lesion with an external "mother-of-pearl" appearance. He called the tumor "tumeur perlée." Although this was not the first description of the lesion, it was the first accurate report. In 1839 Mueller found that many of these tumors contained large masses of cholesterin and because of this he advocated the use of the term "cholesteatoma." Since 1839 this term has been used for an entire group of tumors containing cholesterin, including the cholesteatomas, or cholesteatosis, arising in the bony structures of the cranium from chronic suppuration, the intracranial epidermoids, the pearly tumor of the brain, and the intracranial dermoids. In 1854 von Remak first described the true nature of the tumor. He considered the tumors to be epithelial in structure, arising from misplaced epidermal rests, and classified them as "epidermoids." In 1855 Virchow refuted the use of the term "cholesteatoma" for he felt that this term was applicable to any tumor containing cholesterin and not specific for epidermoid tumors. He classified the lesion as a "Perlgeschwülste." An additional stimulus to the controversy over the origin of epidermoid tumors was given by Perls in 1877, who stated that the tumors were metaplastic and arose from endothelium. The advocates of this theory include Glaeser, Klebs, Nehrkorn, and Kakeshita. In opposition to this theory was Boestrom, who, in 1897, enlarged on the theory of von Remak and presented a clear concise embryologic explanation for the ectodermal inclusions which gave rise to the tumors. He classified the tumor as "piale epidermoide." Another interesting date in the chronology of these tumors is 1856, when the first successful operative procedure for the removal of cholesteatoma was reported by Esmarch. These historical data have been assembled in articles by Rand and Reeves[8] and Critchley and Ferguson[3] wherein the specific references may be found.

The synonyms taken from the literature for the tumors are:

1. Cholesteatoma (Mueller)
2. Pearly tumor.
3. Tumeur perlée (Cruveilheir).
4. Endothelioma (Perls).

From the Army Institute of Pathology, Army Medical Museum, Washington, D. C.

5. Perl krebs.
6. Dermoids.
7. Epidermoids (von Remak).
8. Psammomas.
9. Cutaneous proliferating cysts of brain (Paget).
10. Piale epidermoide (Boestrom).
11. Cholesteatomatous endotheliomata.
12. Epidermoid teratoma.
13. Perlgeschwülste (Virchow).
14. Epidermoid cyst (Love and Kernohan[6]).
15. Meningeal cholesteatoma (Horrax[4]).

Critchley and Ferguson[3] in 1928 expressed the opinion that the term "epidermoid" was best suited to these tumors, and divided them into two types: (1) the epidermoid variety, which consists merely of a layer of stratified squamous epithelium without the skin appendages, and (2) dermoids, which were analogous to the entire layer of skin including the appendages. They considered these tumors to be epithelial neoplasms.

In order to understand clearly the problem of epidermoids, one must review the embryology of the central nervous system. It is well known that the vertebrate nervous system arises from a thickened plate (neural plate) of ectoderm which lies along the mid-dorsal region of the embryo. As development progresses, the neural plate undergoes an infolding, forming the neural groove which has small clusters of cells on either side which form the neural crest. These cells are contiguous with the external layer of ectoderm. As the development progresses further, the neural tube is separated from the neural crest and ectoderm, which now covers the superior surface of the neural tube. It is at this stage of closure that small islands of ectodermal cells may lodge along the neural tube. Through the process of folding and rotation these islands of ectodermal cells usually remain along the base of the adult brain and from these isolated islands of epithelial cells the intracranial epidermoid arises.

The term "epidermoid" is the most suitable for this variety of tumor, for they arise from epiblastic ectodermal rests and may consist of a single layer of epidermis (epidermoids) or the entire thickness of the dermis and epidermis (dermoid). The term "cholesteatoma" should be used with reservation, for many of these tumors do not contain cholesterin or cholesterin clefts.

Only the epidermoids found in the central nervous system will be considered in this paper. Those arising in the cranium have been excluded.

INCIDENCE

Epidermoids are unusual and relatively rare tumors. The diagnosis is seldom made preoperatively and many of them are incidental findings at post mortem. The occurrence varies in different series reported. Tooth,* in 1913, in a series of 258 cases of brain tumor, found only one epidermoid, representing 0.4 per cent of the entire group. Bailey,* in 1920, recorded 2 cases in a series of 550 verified brain tumors in the Cushing Clinic, or a percentage of 0.37. Mahoney,[7] in 1936, collected the information on central nervous system epider-

*Cited by Rand and Reeves.[5]

moid tumors in the literature. He accumulated 112 intracranial epidermoids. He also stated that 15 epidermoid tumors were found in the 2,500 verified intracranial tumors in this later Cushing series. This represents 0.6 per cent of all intracranial neoplasms. Of the 518 brain tumors studied at the Army Institute of Pathology, 5 cases of intracranial epidermoids were recorded from Pearl Harbor to V-J day. This represents 0.96 per cent of the intracranial neoplasms. Four cases of epidermoid cysts of the skull were recorded over the same period but will be omitted from further discussion in this paper.

LOCATION

A majority of the authors have included the diploic and spinal epidermoids as well as the cerebral epidermoids in their statistics. We are limiting our discussion to the central nervous system lesions. The tumors may be found anywhere in the cerebrum but are most frequently found along the base of the brain. The favorite location is in the region of the third and fourth ventricles and the cerebellopontile angle. The cases reported by Mahoney[7] were divided as follows:

Diploic	23
Intraspinal	7
Fourth ventricle	15
Parapituitary	44
Parapontine	53
Total	142
Intracranial	112

The epidermoid cysts may extend into the surrounding pia and bony structures. This extension is by pressure erosion of the pia, dura, and bone rather than by invasion. The 3 cases of epidermoid of the central nervous system were located in the suprasellar region, in the third ventricle, and in the left occipital lobe. The remaining 2 cases were found in the choroid plexus. The lesion in the left occipital lobe was on the inferior surface and had become adherent to the dura and eroded the occipital bone. The other lesions compressed the surrounding structures, but did not invade them.

Of the 5 cases the two incidental lesions were found in the choroid plexus. One of these lesions measures 3 by 5 by 2 cm., while the other measures 1.5 cm. in diameter. The cross section revealed the choroid plexus to contain a yellow-brown soft puttylike material. On removing this material, small papillary excrescences were visible. As has been previously stated, many men feel that this lesion in the choroid plexus is an example of metaplasia of endothelial cells. One can postulate that epithelial nests may be incorporated in the choroid plexus as well as elsewhere in the brain, during the embryonic development, because of the close proximity of this area with the ectoderm.

PATHOLOGY

The origin of these tumors has been discussed previously. The tumors are usually well encapsulated and easily separated from the surrounding brain tissue. The point of origin is usually discernible. The capsule is pearly gray and often

has the glistening appearance of "mother-of-pearl." A few compressed blood vessels are present over the surface. The surrounding brain tissue is compressed and a few small hemorrhages may be seen. The pia and dura may be firmly attached to the external surface. In one of the tumors the dura was incorporated, so that it gave the appearance that this tumor might have arisen from the dura. The tumor mass is firm and resilient. The surface made by sectioning presents a thin fibrous silver-gray wall. The majority of the tumor consists of a space filled with a thick puttylike tan-brown material which can easily be removed from the capsule. The internal lining is smooth and glistening with an occasional papillary excrescence. Occasionally the papillary folds form larger polypoid masses (Fig. 1). If skin appendages are present in the wall, the cystic tumor contains hair and sebaceous material in the central cavity. Cholesterol crystals were seen in 3 of the cases, characterized by irregular shimmering crystals intermingled throughout the soft puttylike material. Flecks of calcium may be imbedded in the fibrous connective tissue capsule.



Fig. 1.—A suprasellar epidermoid tumor (cyst) lying immediately beneath the base of frontal lobes and projecting into the basal portion of the longitudinal fissure. Note the thin fibrous wall with intracystic papillary projections. The contents of the cyst have been removed. (A.I.P. Acc. No. 118013.)

MICROSCOPIC

The microscopic appearance of the wall in all of these cases was that of a narrow zone of stratified squamous epithelium. This layer of stratified squamous

epithelium lies on a relatively avascular zone of connective tissue. In some areas the vessels are small and thin walled, and there was evidence of a chronic inflammatory reaction characterized by a few lymphocytes and focal fibrous scars. The basal layer of the stratified squamous epithelium consists of pavement type of cells with elongated basophilic nuclei. The zone immediately above resembles the prickle cell zone of the epidermis. The cells are irregular in shape with an eccentric nucleus and are separated from one another by numerous intercellular bridges. These cells contain keratohyaline granules. The inner zone is made up of desquamated cornified epithelial cells which make up the bulk of the contents of the cyst (Fig. 2). A few degenerating nuclei are seen in desquamated material. Cholesterin may also be present. A few cholesterol clefts in the foreign body giant cells are frequently seen. If the skin appendages are present, sebaceous glands, sweat glands, and hair follicles may be present beneath the layer of stratified squamous epithelium. King[5] has described three specific distinct layers to the wall of the epidermoids. The first being an outer, relatively avascular connective tissue capsule which varies in thickness and is the supporting connective tissue for the second layer, which consists of the zone of stratified squamous epithelium. The third, or internal, layer consists chiefly of cornified epithelium and epithelial debris.

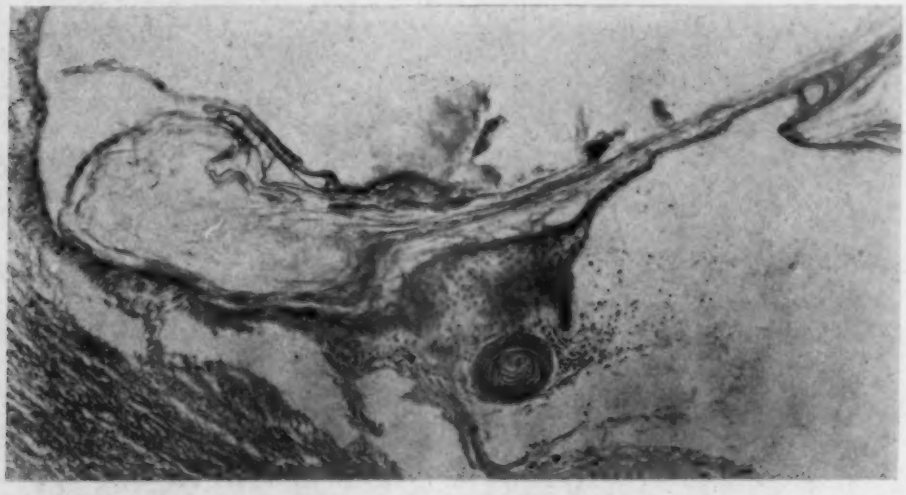


Fig. 2.—The wall of the epidermoid tumor (cyst) consists of a layer of stratified squamous epithelium lying on a connective tissue capsule. The cyst is filled with cornified desquamated epithelial cells and debris. Note the small so-called "daughter cyst" filled with keratohyaline material lying in the adjacent connective tissue. (A.I.P. Acc. No. 65044.)

DIAGNOSIS

The clinical syndrome is not specific and is usually characterized by the signs of an increased intracranial pressure secondary to the presence of the tumor. The diagnosis of intracranial neoplasm is usually made. The tumor may be localized by x-ray if the bone of the calvarium is involved or an adequate amount of calcium is present in the capsule to give rise to a shadow on the x-ray film. Encephalography and ventriculography may aid in the location

of an intracerebral epidermoid. The presenting symptoms in the cases reviewed vary from severe focal headaches to a sudden onset of coma. In two of the cases no symptoms were present and the lesions were incidental findings at the time of autopsy. The other three cases presented papilledema in varying degrees and all were associated with blurring of vision. Other symptoms and signs depended on the location of the tumor. Craniotomies were done in three cases. At the time of the writing of this paper all were in good health following the surgery. Though the symptom complex is not specific and a preoperative diagnosis is difficult, the specific diagnosis of epidermoid is readily made at the time of operation because of the pearly appearance of the tumor. The tumors are congenital and must attain an adequate size to compress the surrounding brain or give rise to increased intracranial pressure by causing obstruction of the ventricular system before symptoms appear; thus the duration of the lesion cannot be accurately determined. In the three cases diagnosed as brain tumors prior to operation, the duration of symptoms varied from four weeks to one and a half years. The lesions occurred in individuals who were 19, 33, and 48 years of age, respectively.

DISCUSSION

The presence of epidermoid cysts in the cerebrum is a rare occurrence. The pathogenesis of such a lesion probably is the result of epithelial inclusions from the ectoderm which approximates the neural tube during the process of closure. The close proximity of the ectoderm to the neural tube has been demonstrated by Globus* and others. He states that the epidermis and meningeal structures are in close approximation early in the development of the nervous system and at such a time clusters of primordial epidermal epithelial elements may be incorporated in the matrix of the pia and dura. An occasional lesion, especially those found in the choroid plexus, may arise from metaplasia of endothelial elements. The choroid plexus is a direct derivative from the medullary epithelium which is ectodermal in origin, and embryonic ectodermal inclusion may give rise to tumor formation, occurring in these nests after the lapse of considerable time. The presence or absence of skin appendages, that is, sweat glands, sebaceous glands, and hair follicles, has resulted in the division of the tumors into epidermoids and dermoids. The epidermoid tumors or cysts are those containing only the stratified squamous epithelium, while the dermoid tumors or cysts are made up of all layers of the epidermis and dermis. All five cases represent the epidermoid type of tumor. Cholesterin was found in three of the cases. The common usage for the term "cholesteatoma" should be dropped, as suggested by Virchow, for many do not contain cholesterol.

It is interesting to note the similarity of the intracerebral epidermoids with the cholesteatomas (epidermoids) occurring in the petrous portion of the temporal bones. These lesions are invariably secondary to chronic suppuration. The small islands of epidermis which are isolated in the bone due to the inflammatory process are the site of origin of these lesions. A majority contain masses of cholesterin, as well as cornified stratified squamous epithelium and debris.

*Cited by Rosenstein.[39]

The lesions of the temporal bone may extend into the dura and pia arachnoid. Signs of increased intracranial pressure due to involvement of brain may be present. A history of previous ear disease or mastoiditis usually accompanies the signs of increased intracranial pressure. Microscopically, the stratified squamous epithelium resembles the epithelium of the epidermoids of the central nervous system except that the chronic inflammatory reaction and fibrosis are more prominent. The contents of the cysts are similar. These lesions are also called cholesteatomas or cholesteatosis of the petrous bone secondary to chronic otitis media.

SUMMARY

Five cases of epidermoid tumors of the central nervous system have been presented. None of these lesions showed skin appendages in the microscopic sections. The pathogenesis of epidermoid tumors has been discussed. It is felt that the term cholesteatoma should not be used in the classification of these tumors. The term epidermoid or dermoid is preferred. The epidermoid tumor represents an epithelial neoplasm, tracing its origin to an embryonic remnant of epidermal cells during the development of the neural tube.

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The American Academy of Oral Pathology

Announcement is made of the organization of The American Academy of Oral Pathology, devoted to the interests of those qualified in the field of oral pathology. This Academy was organized through the efforts of the Registry of Dental and Oral Pathology of the American Dental Association, Army Institute of Pathology, Washington, D. C.

Any dentist in good standing in the American Dental Association may request membership, provided he has had some advanced training in clinical, morphologic, or experimental oral pathology, and is endorsed by two Fellows of the Academy.



Left to right: Dr. Lester A. Cahn, President-Elect; Dr. J. R. Blayney, Second Vice-President; Dr. Donald Kerr, First Vice-President; Dr. H. B. G. Robinson, Editor; Lt. Col. J. L. Bernier, Secretary-Treasurer; Dr. Kurt Thoma, President; Dr. Henry Swanson, Chairman, Museum Committee.

Elevation to Fellowship in the Academy is by examination. Candidates for Fellowship must, after attaining membership, submit evidence of training above the undergraduate training level, such as postgraduate study, teaching, research activities, or active association with a department of pathology of an acceptable institution. Such candidates will be certified to examination only after careful scrutiny by the Fellowship Committee and the Executive Committee. Successful candidates shall be elected Fellows of The American Academy of Oral Pathology and awarded a suitable certificate.

The first meeting of the Executive Committee of The American Academy of Oral Pathology was held on June 7, 1946, at the Hotel Statler, Washington, D. C. The following are the temporary officers who, at present, constitute the Executive Committee:

Dr. Kurt H. Thoma, President.
Dr. Lester A. Cahn, President-elect.
Dr. Donald Kerr, First Vice-President.
Dr. J. R. Blayney, Second Vice-President.
Dr. H. B. G. Robinson, Editor.
Lt. Col. J. L. Bernier, Secretary-Treasurer.

At this meeting the plans of organization were completed, and Charter Fellows were selected. Individuals chosen to Charter Fellowship were those who, in a selected preliminary survey, had indicated active interest in The American Academy of Oral Pathology.

The first annual meeting of The American Academy of Oral Pathology will be held in Chicago, preceding the midwinter meeting of the Chicago Dental Society in February, 1947. Full details regarding this meeting will be announced later.

Further information relative to Membership may be obtained by addressing Lt. Col. Joseph L. Bernier, Secretary-Treasurer, Army Institute of Pathology, Washington 25, D. C.



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William Thomas Green Morton

William T. G. Morton, a Boston dentist, born in Charlton, Massachusetts, Aug. 19, 1819, demonstrated Surgical Anesthesia at the Massachusetts General Hospital on Oct. 16, 1846. The effect of this demonstration caused a development of the science of anesthesia, "anesthesia" being a word coined by Dr. Oliver Wendell Holmes. It is derived from the Greek, meaning "a state without pain."

This humane act, which today is taken for granted when an operation is performed, gave rise to extensive research and an endless chain of investigation, through which humanity has been given the modern methods of anesthesia. An obscure man thus made himself immortal by introducing a long-needed aid to surgery. But Morton's discovery did not end with these improvements; it brought with it great developments in the field of surgery, undreamed of only a few decades ago.

Let us all do honor to our fellow practitioner, whose statue stands in the Smithsonian Institution and who was elected to the Hall of Fame of New York University in 1920, a peer among men who have devoted their lives to the conquest of pain.

The War Issue

A REPORT OF THE DENTAL DEPARTMENT OF THE 6TH GENERAL HOSPITAL ORGANIZED FROM THE DENTAL DEPARTMENT, MASSACHUSETTS GENERAL HOSPITAL

COLONEL ROBERT G. RAE, D.C.,* MAJOR SOMERS H. STURGIS, M.C.,† AND MAJOR DANIEL J. HOLLAND, JR., D.C.‡
WITH AN INTRODUCTION BY KURT H. THOMA, D.M.D.§

THE MASSACHUSETTS GENERAL HOSPITAL DENTAL DEPARTMENT DURING WORLD WAR II

KURT H. THOMA, D.M.D.

OF THE thirty-eight members of the Hospital's Dental Department, eleven served in the Armed Forces during World War II. Those who stayed at home for various reasons did their best to carry on under the additional burdens placed upon them, both in the clinic as well as by discharging additional duties in various civic affairs.

This issue is devoted to a report from the men who have served with the 6th General Hospital, staffed by members from the Massachusetts General Hospital.

Those who had other military assignments should not be forgotten, however, and this introduction should serve to give credit to all for their achievements and sacrifices.

The following were members of the 6th General Hospital:

Colonel Robert G. Rae, Chief of the Dental Department of the 6th General Hospital, who, after serving nine months at Camp Blanding, Florida, saw two and one-half years of foreign duty in Africa and Italy. Activated in the grade of Major, he was raised to the rank of Colonel in recognition of his administrative and professional abilities.

Major Daniel J. Holland, Jr., who was assigned to take the maxillofacial course at the Army Medical School in Washington, D. C., became chief of the Oral Surgical Section of the 6th General Hospital and, with the surgical members of the maxillo team, surveyed the facial casualties in the hospitals of the North African Area, outlining policies in regard to treatment and

*Dental Surgeon, Massachusetts General Hospital.
†Assistant Surgeon, Massachusetts General Hospital.
‡Assistant Dental Surgeon, Massachusetts General Hospital.
§Oral Surgeon and Chief of Dental Department, Massachusetts General Hospital.

evacuation. Major Holland was dental historian of the African and Mediterranean areas and was awarded the Bronze Star Medal, Order of the Crown (Italy), War Merit Cross, and the Military Order of St. George. He returned to the Massachusetts General Hospital after the termination of the War to serve as Resident in Oral Surgery.

Also in connection with the 6th General Hospital was *Major H. B. Seyfarth,* who was in charge of the prosthetic section. After his duty in Camp Blanding, he saw active temporary duty with the United States 5th Army in North Italy, when he brought dental laboratory facilities to combat troops. His skill and devotion to duty provoked great admiration and respect among the military personnel.

Lt. Colonel F. K. Poulin, activated as First Lieutenant with the 6th General Hospital, was transferred to the 160th Station Hospital, destination England, and later transferred to the 12th Air Force. He saw duty in the North African and Italian campaigns. He was appointed Staff Dental Officer in the Office of the Air Surgeon in 1944, to act as Assistant to the Surgeon, to coordinate the dental service throughout the 12th Air Force. He received the Bronze Star Medal for meritorious service in direct support of combat operations and was awarded five battle stars.

Several members of the Dental Department of the Massachusetts General Hospital served in other units.

Lt. Colonel M. S. Strock was Chief of the Dental Service of the 5th General Hospital. He saw foreign service from early 1942 in Ireland, Scotland, England, and France.

Lt. Colonel Henry Carney was a reserve officer in the Army since World War I. He was in the convoy which invaded North Africa on November 8, 1942, landing with the Central Task Force. He saw a good deal of action in the Tunisian Campaign with the 148th Surgical Hospital and was later assigned to the 12th General Hospital, 2nd Convalescent Hospital, and still later as Chief of the Dental Service to the 52nd Station Hospital in Naples, which was designated as a maxillofacial center. For his work he was awarded the "Legion of Merit." Later, he was appointed Chief of Dental Service of the 64th General Hospital in Leghorn, Italy. An outstanding accomplishment of his military service was the invention of a "Foreign Body Detector," "a traction appliance for cervical vertebra fractures," as well as a number of other instruments and mechanical devices for the Army Medical Department.

The Navy was well represented by the following men:

Commander F. I. Rhodes, commissioned Assistant Dental Surgeon in 1924 with the rank of Lieutenant (j.g.), became Senior Dental Officer and Oral Surgeon, United States Naval Hospital, Chelsea, Massachusetts, in 1939, where, under his direction, the dental service was expanded and enlarged to provide for the increased number of war casualties. He became Senior Dental Officer, U.S.S. Denebola in 1942, and later of Fleet Hospital No. 116, Camp Parks, Shoemaker, California, and subsequently returned to the Naval Hospital, Chelsea, Massachusetts.

Commander Joseph A. Doherty served with the Navy in the Southwest Pacific for three and one half years. He was attached to a mobile hospital as Senior Officer in charge of maxillofacial surgery.

Lt. Commander H. E. Dyer was Area Senior Dental Officer, United States Navy, stationed at Sampson Naval Station, then transferred to Naval Proving Ground in Dahlgren, Virginia.

Lt. Commander Keith Shangraw was first stationed at Quonsett Naval Station in 1943, attached to Fast Carrier Force in the Pacific Theater for twenty-two months. He was Senior Dental Officer on the Carrier U.S.S. San Jacinto which received the Presidential Citation for courageous naval participation.

Lt. Commander George M. Noss was activated as Lieutenant (j.g.). He was District Dental Officer, U. S. Coast Guard, 1st Naval District, promoted to Lieutenant in 1944, and subsequently to Lieutenant Commander, accomplishing a large volume of prosthetic dentistry of high caliber.

Lieutenant F. B. Greene joined the United States Naval Reserve and was stationed at United States Naval Training Station at Newport, Rhode Island. Subsequent changes were to the Amphibious Training Base at Camp Bradford, Virginia, and the Naval Separation Center at Lido Beach, New York.

Twice within twenty-five years, the Massachusetts General Hospital has sent forth its staff members to render succor and aid in time of war. The Massachusetts General Hospital dental members serving in various organizations in the four corners of the globe contributed generously to the cause and fulfilled admirably the high traditions of the parent institution.

THE 6TH GENERAL HOSPITAL
(MASSACHUSETTS GENERAL HOSPITAL UNIT)

ROBERT G. RAE, D.M.D.*

IN 1917, under the auspices of the American Red Cross, a number of large hospitals throughout the country sponsored Base Hospital Units to augment the medical program of the United States Army. The Massachusetts General Hospital sponsored Base Hospital No. 6, which was called into active service and sailed overseas in the summer of 1917. The unit set up station in a suburb of Bordeaux, France, and had a distinguished record of service with the American Expeditionary Force during World War I.

Twenty-five years later, the Massachusetts General Hospital again responded to the country in time of stress and from its professional staffs organized the 6th General Hospital. Called to active duty on May 15, 1942, a nine-month period of training and waiting at Camp Blanding, Florida, followed. During this period, the hospital's dental service actively participated in the oral rehabilitation of the large numbers of troops being prepared for overseas duty, and became familiar with, and adjusted to, the policies and regulations of the Army Dental Corps. At the time of activation the dental service comprised:

Major Robert G. Rae	Chief of Service
Captain Joseph H. DeMers	Oral Surgeon
Captain Daniel J. Holland, Jr.	Oral Surgeon
Captain Hermann B. F. Seyfarth	Prosthetist
Lieutenant Robert M. Bailey, III	Operative Section
Lieutenant Calvin R. Coggins	Operative Section
Lieutenant Frederick K. Poulin	Operative Section

In addition to the seven officers, the dental component of the unit included two female civilian hygienists and seventeen enlisted men.

Before sailing for overseas duty the dental group was reduced to five officers (Lieutenants Poulin and Coggins being transferred to other organizations), and the hygienists were dropped from the allotment of authorized personnel. The maxillofacial team of Captain Somers H. Sturgis, M.C., and Captain Daniel J. Holland, Jr., D.C., attended the course for maxillofacial and plastic surgery at the Army Medical Center, Washington, D. C.

The unit arrived in Casablanca, French Morocco, North Africa, three months after the invasion and remained for fifteen months. Handicapped to some extent by lack of supplies, complete dental health service was extended not only to the patients confined to the hospital but, on an outpatient status, to troops stationed near by.

*Colonel, Dental Corps (inactive); Chief of Dental Service, 6th General Hospital; Dental Surgeon, Massachusetts General Hospital.

The 6th General Hospital was the last hospital in the chain of evacuation from North Africa, and the battle casualties had been through one or more hospitals before arriving at the 6th. However, regardless of the distance from the active front, the dental clinic remained one of the busiest spots in the hospital and the demands for dental treatment—particularly prosthesis—were heavy. Under the supervision of Major Seyfarth, the laboratory technicians accomplished a large amount of prosthetic dentistry.

Every patient in the hospital, when ambulatory, came to the dental clinic for examination, and an effort was made to complete all required dental treatment before the patient was discharged or transferred. In some instances when large numbers of patients were admitted for a brief hospital stay, before being evacuated to the United States, definitive dental care was not possible.

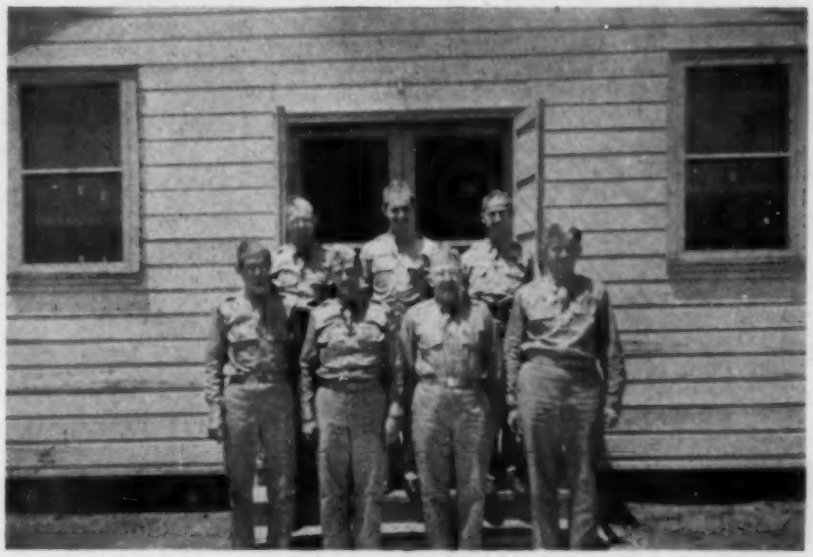


Fig. 1.—(Left to right). Front row: Bailey, Poulin, Rae, Coggins. Back row: Seyfarth, Holland, DeMers.

In the fall of 1943, the maxillofacial team surveyed and examined 342 maxillofacial casualties in fifteen American military hospitals and submitted a detailed report of their findings to the Surgeon of the North African Theater of Operations. Later, they were instrumental in outlining the policy of treatment for jaw and face casualties in the forward and rear echelons.

During the stay in Casablanca, there were changes in both the officer and enlisted dental personnel; the problem of dental supplies was corrected; and the service ran at peak capacity.

The mission in Casablanca ended on May 16, 1944, when personnel and equipment were moved by troop trains to Oran, Algeria. By hospital ship the unit crossed the Mediterranean and landed in Naples, Italy, early in June. Shortly after the Eternal City fell to Allied control, the 6th General Hospital entered Rome and took over a large, rambling convent consisting of nine court-

yards, many spires, plus a chapel and auditorium. The Germans had previously requisitioned the building as a military hospital and in haste had retreated just prior to the entrance of Allied troops. The building was in a discouraging state of defilement and the entire hospital staff was challenged to restore American standards of sanitation and inspection.



Fig. 2.—A large girls' school in the center of Casablanca taken over by the 6th General Hospital.

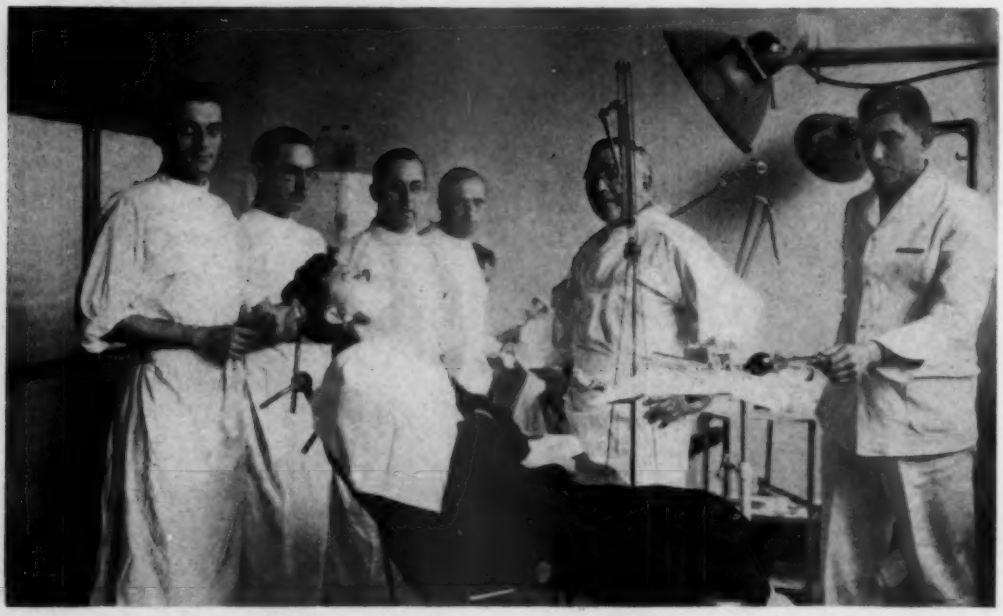


Fig. 3.—A soldier's dental status being reviewed and discussed by the Chief of Service. (Left to right). Lieutenant Bailey, Captain Holland, Captain DeMers, Captain Seyfarth, Major Rae, Tec. 5th Grade Tyman.

In contrast to Casablanca, the 6th was now the first general hospital in the line—with the active front a short distance north of Rome. Battle casualties were received directly from the forward areas, and although equipped as a 1,500-bed hospital, our patient census fluctuated between 2,000 and 2,500. The Rome experience was short-lived but busy.



Fig. 4.—Main entrance to the 6th General Hospital in Rome.

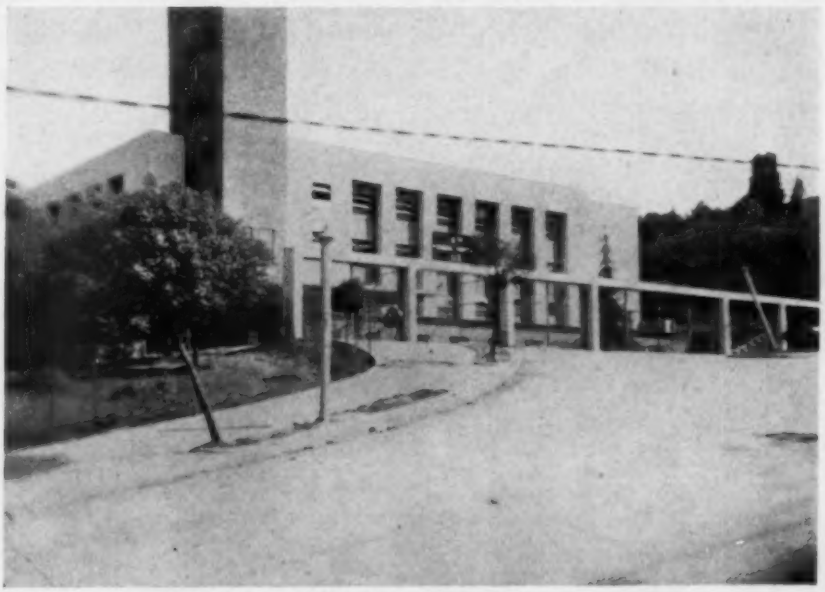


Fig. 5.—Front entrance to hospital at Bologna, Italy.

A 30-bed maxillofacial ward was organized as part of the dental service and some of the most critically ill and seriously wounded facial battle casualties were treated by the maxillofacial team during this phase of the war.

The advances northward made by the American 5th Army soon found Rome far to the rear, and in December, 1944, the 6th General Hospital closed. For the next four months the hospital personnel were placed on temporary duty with other medical installations throughout northern Italy. May 1, 1945, found the hospital setting up station in Bologna. A large modern building, formerly used as a school of engineering, had been selected for the new site.

The announcement of the end of hostilities in Italy on May 8 did not halt the work of expanding the new hospital. The wards were soon filled with an equal number of American and German casualties. Mixed in with the "Kraut" patients were Italians, Poles, Russians, and other nationalities who had been forced to serve the Nazis. These foreign patients reflected the chaos of the German medical program toward the end of the war, and when the obvious lack of dental care of the Germans and Italians was viewed, one could not help but feel that the American Dental Corps had done a fine job in caring for the dental health of the U. S. troops.

In July, 1945, the hospital was closed and the task of demobilization under the point system was begun.

The majority of the 6th General Hospital personnel arrived back in the United States in late September, 1945, after 31 months of foreign service, during which time 28,966 patients were hospitalized.

The Dental Service admitted 11,447 patients for clinic treatment and offered complete dental health service.

As a branch of the Medical Department whose aim was to preserve the fighting strength of the Armed Forces, dentistry actively participated. The maintenance and preservation of sound oral health, the rehabilitation of dental "cripples," and the dental contribution in restoring function and esthetics in those wounded about the face and jaws, was an integral share of the military mission of the 6th General Hospital.

OBSERVATIONS ON 200 JAW FRACTURE CASES ADMITTED TO THE 6TH GENERAL HOSPITAL

MAJOR SOMERS H. STURGIS, M.C., AND MAJOR DANIEL J. HOLLAND, JR., D.C.

INJURIES to the face resulting in fracture of one or both jawbones were infrequently admitted to a General Hospital for initial or primary treatment. Maxillofacial battle casualties wounded in action usually received first aid on the spot and soon were transported to field or evacuation hospitals where important resuscitative measures and early definitive treatment were instituted. In the chain of evacuation these patients arrived at general hospitals where final treatment was rendered and the patients discharged to duty, or, because of severe injury requiring prolonged hospitalization or because of permanent disability, they were evacuated to the United States. Likewise, jaws fractured in accidental injuries unassociated with combat were usually treated at near-by station hospitals, and only when special care was required were they admitted to a general hospital.

The following summations are from 200 patients with jaw fractures—battle casualties and accidental injuries—admitted to the 6th General Hospital overseas between March, 1943, and June, 1945.

TABLE I

NUMBER OF PATIENTS WITH JAW FRACTURES		200
Accidental injuries	96	
Battle casualties	104	

ACCIDENTAL INJURIES

The nonbattle casualties or accidental injuries resulting in jaw fractures were not unlike those seen in civilian life. As a rule, these were uncomplicated fractures in which there was no loss of bone and the soft tissue damage was minimal. Unless complicated by other injuries, these patients were returned to military duty within eight weeks and offered no special problems in management. The etiology of the 96 jaw fractures caused by accident is given in Table II.

TABLE II

"Brawls" or fights	33	34%
Jeep accidents	19	20%
Duty accidents*	44	46%

*Falls, airplane crashes, sports, etc.

The jeep cases were frequently seriously injured but without marked injury to the soft tissues of the face. The fist fight cases were, as expected, uncomplicated fractures compounded intraorally and with no soft tissue loss.

Maxillofacial team, 6th General Hospital.

The following case histories are representative of the three main classifications of military accidental jaw fractures.

1. *Brawls or Fights.*—Case No. 26175. A 21-year-old soldier was admitted to the 6th General Hospital in Rome on Dec. 4, 1944. Six weeks previously he had been struck by an Italian civilian in a brawl and taken to a near-by military hospital, where a diagnosis of bilateral compound fracture of the mandible was made. Continuous loop wiring was applied. Four weeks later, at the same hospital, because of considerable upward displacement of the distal segment, a 1 inch incision was made behind the left angle. A double strand of brass ligature wire was passed through a drill hole at the lower border and attached to a plaster head cap by elastic traction—pulling downward and laterally, as shown in Fig. 1.



Fig. 1.—Outward and downward traction on ramus segment by means of interosseous wire attached to rod from plaster head cap.

On admission to our hospital six weeks after injury, the intraoral wires were loose, the mouth hygiene was extremely poor, there was intraoral suppuration from around the last molar tooth on the lower left and the second premolar on the lower right, both of which were involved in the fracture lines, and there was suppuration around the extraoral wire at the left angle (Figs. 2*A*, 2*B*, and 2*C*).

The plaster head cap, interosseous wire, and traction were removed; the wires were taken off the teeth, and the two teeth in the lines of fracture were extracted. The oral hygiene was improved and the septic skin wound at the angle was treated. (Fig. 3.)

Four days after admission, intermaxillary Ivy loops were applied and under local anesthesia an open reduction by direct transosseous wiring was performed. An anatomic approximation of the segments on the left side was obtained. (Figs. 4*A* and 4*B*.)

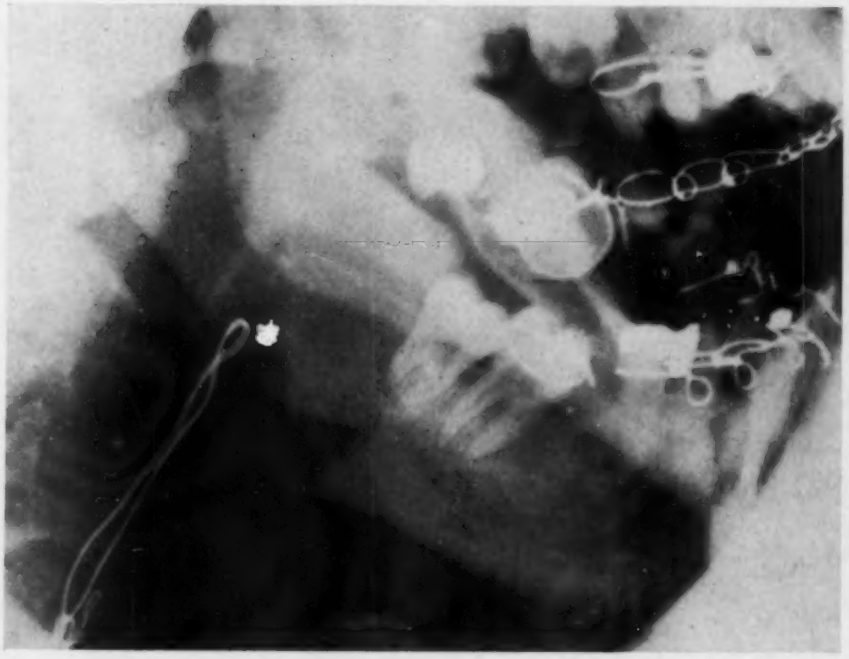


Fig. 2*A*.—Left lateral jaw showing upward displacement of distal segment and double strand of brass wire through drill hole at lower border.



Fig. 2*B*.—Right lateral jaw showing fracture in second premolar region with some upward displacement. The second premolar has been extracted.

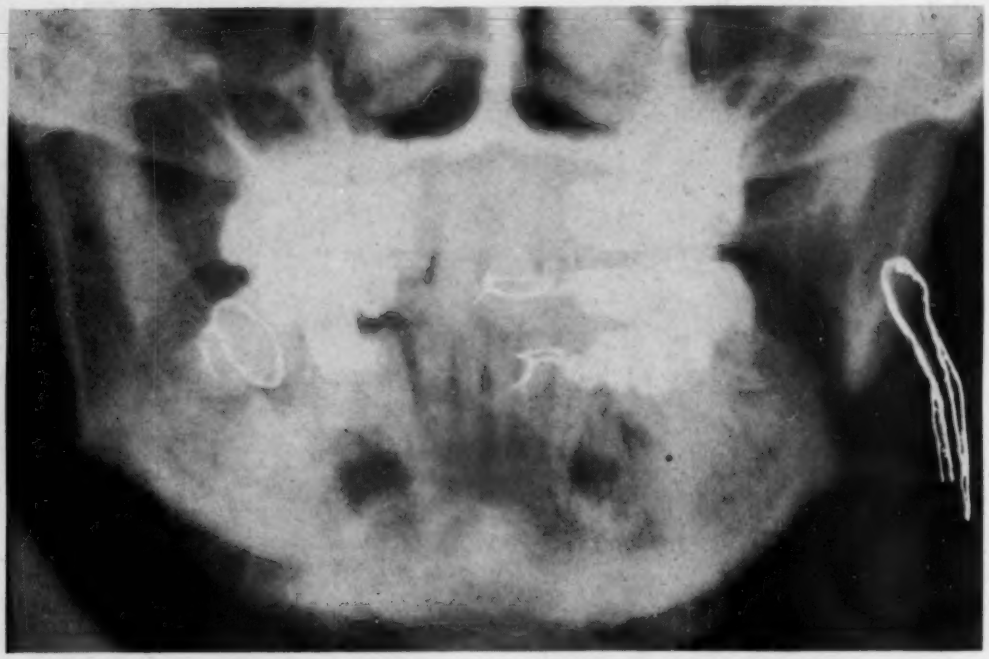


Fig. 2C.—Anterior view showing separation of the fragments at the left angle six weeks after injury (wire retraced).



Fig. 3.—Left lateral x-ray with interosseous wire removed and third molar tooth in fracture line extracted.

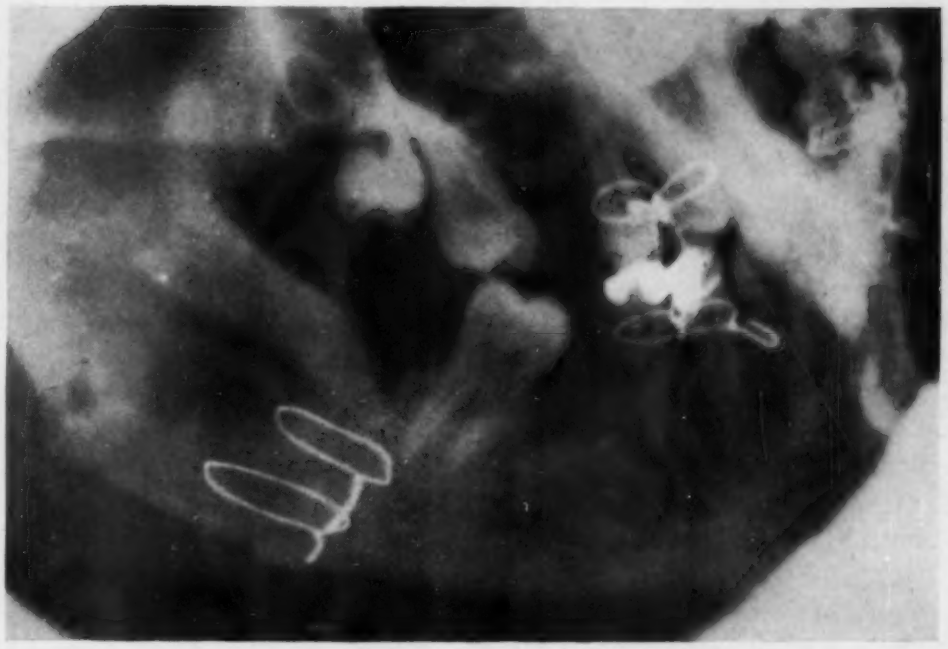


Fig. 4*A*.—Left lateral x-ray showing anatomic approximation following open reduction and direct transosseous wiring.

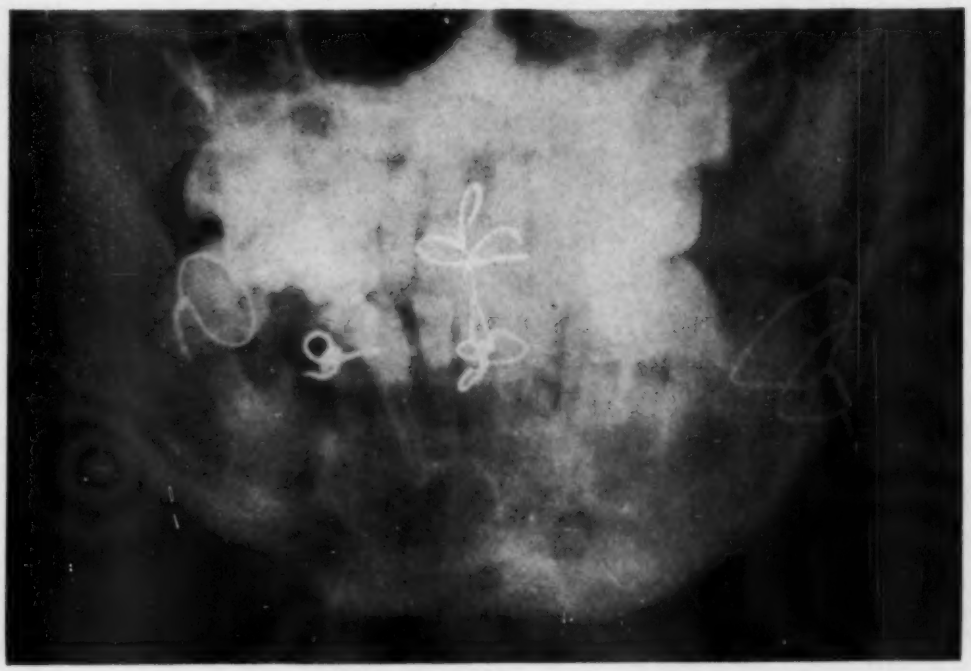


Fig. 4*B*.—Anterior view after reduction. Compare with Fig. 2*C*.

Although the segments on the right side were not in optimal position, seven weeks had elapsed since injury and union was well under way, so it was decided not to disturb the callus. (See Fig. 2*B*.)

Six weeks after the direct wiring, there was firm union on both sides, the patient was eating a regular diet and was discharged to full military duty. The transosseous wires were not removed.

Comment.—This bilateral fracture of the mandible was originally poorly treated at a previous installation, as follows:

1. Teeth were left in the lines of fracture, which were not essential to reduction or fixation and which subsequently contributed to infection in the fracture lines.

2. After the original intermaxillary wiring, it was observed that the distal segment at the left angle was markedly displaced. One month elapsed before attempting to improve the position.

3. The wire, passed through the lower border and then attached to a plaster head cap by elastic traction, kept the segments separated. (See Fig. 2*C*.)

4. The intraoral wires were loose, and there had been no instruction or supervision of oral hygiene.

The open reduction operation by direct transosseous wiring gave excellent results.

2. *Jeep Accidents.*—Case No. 25970. A 37-year-old soldier was admitted to the 6th General Hospital on Nov. 6, 1944, from a near-by station hospital, twelve hours after having been involved in a jeep accident. The jeep in which he was a passenger left the road and overturned, killing the driver and seriously injuring the patient.

On admission the patient was in shock. There were deep lacerations over both eyes, multiple abrasions of the face and scalp, and an obvious compound fracture of the mandible in the symphysis region. The maxilla was freely movable and was displaced downward and distally. The upper anterior teeth were partly avulsed from their sockets. There was bleeding from the mouth and cerebrospinal rhinorrhea. The middle third of the face was depressed.

The patient responded well to resuscitative measures, and five days after admission the nasal cerebrospinal drip had ceased and a neurological examination was essentially negative.

X-rays revealed, besides the fracture of the mandibular symphysis, comminuted fractures of the maxilla, fracture of the nasal bones and separation of the nasofrontal suture extending into the ethmoids, as shown in Fig. 5.

The mandibular fracture was reduced by means of a labial arch wire ligated to the teeth, as shown in Fig. 6.

The nasal bones were reduced and the maxilla was manually manipulated and brought forward and elevated by means of stainless steel wires attached to the right and left molars, brought externally through the cheeks and attached by rubber traction to rods from a plaster head cap. (Figs. 7 and 8.)



Fig. 5.—Arrows point to fracture line and separation of nasofrontal suture and horizontal fracture through lateral body of maxilla.

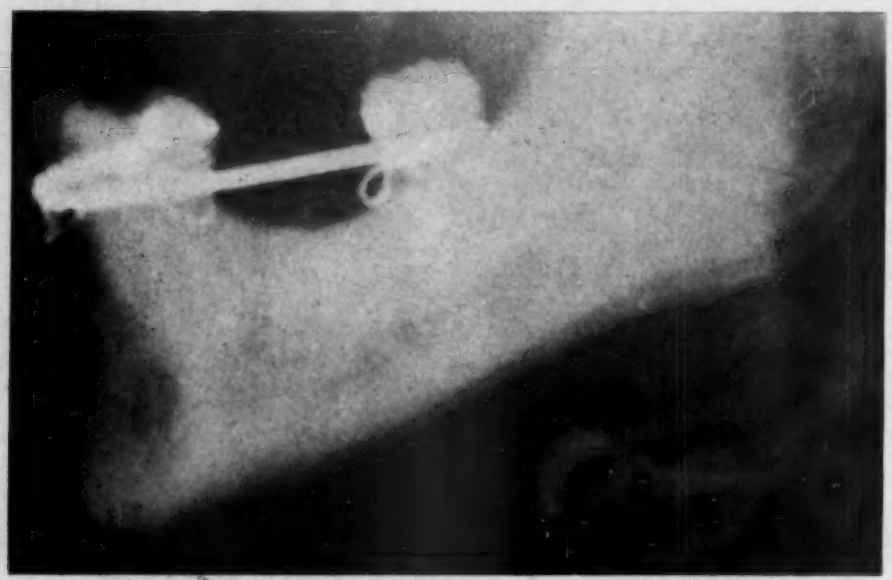


Fig. 6.—Labial arch band used to reduce mandibular fracture.



A. ***B.***

Fig. 7.—*A* and *B*. Photographs showing wires from the molar teeth, through the cheeks and connected to plaster head cap.

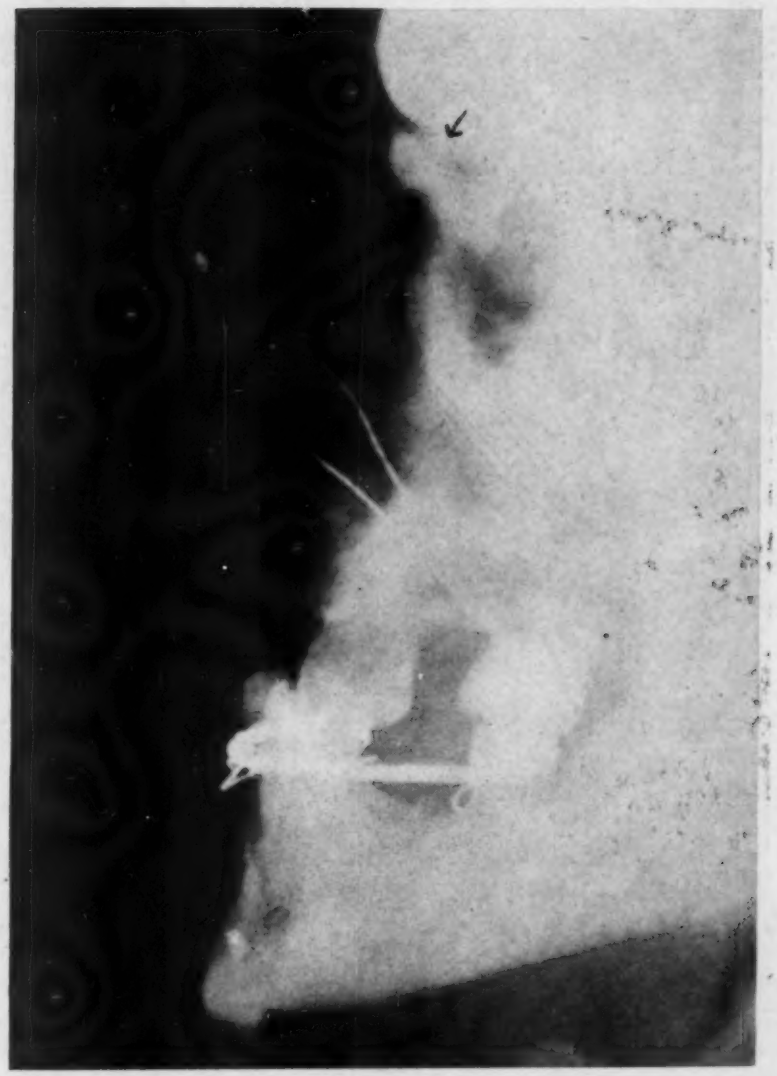


Fig. 8.—Lateral x-ray showing direction of wires. Note reduction in nasofrontal separation.

In twelve days the separation of the nasofrontal suture was reduced and the maxilla was in good position. The loose, partly avulsed maxillary teeth were removed, and nine weeks after injury the mandibular wiring was removed and there was firm union. Later, a full upper denture and lower partial denture were constructed, and the patient was discharged to duty.

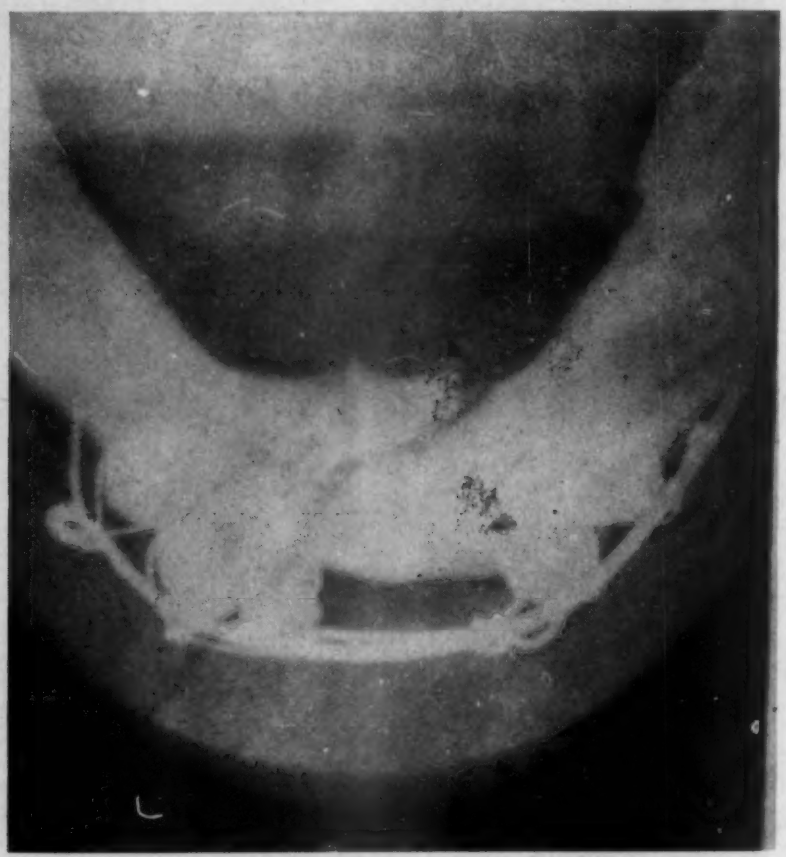


Fig. 9.—Symphysis fracture after reduction.

Comment.—The oblique fracture through the symphysis was not in perfect alignment, as shown in Fig. 9, and perhaps circumferential wiring would have bettered the coaptation. The multiple fractures of the maxilla were reduced when the maxilla was still mobile and were held in satisfactory relation by wires running from the molar teeth out through the cheeks and attached by traction to a plaster head cap. The end result was very satisfactory.

3. *Duty Accidents.*—Case No. 14125. A 36-year-old corporal fell into a 35-foot stone quarry on Jan. 13, 1944, and received fractures of the right clavicle, right wrist, right zygoma, and bilateral compound fractures of the edentulous mandible. In addition to multiple abrasions and contusions, he also sustained a through-and-through laceration of the right cheek. At a near-by hospital he was treated for shock, the soft tissue wounds were dressed, and the fractures of the right wrist and right clavicle were reduced. An attempt was made to insert the

patient's lower denture as a splint, but because of the deformity of the mandibular ridges the attempt was unsuccessful.

Seven days after injury he was admitted to the 6th General Hospital. The fracture of the right zygoma was palpable, as was the depression of the right infraorbital ridge. The fractured segments of the mandible were freely movable, and there was an intraoral projection and overriding of the displaced fracture on the right side. (Fig. 10.)



Fig. 10.—Right lateral jaw x-ray showing upward displacement of the distal fragment. The sharp edge projected through the oral mucosa into the mouth.

Four days after admission the zygoma was elevated by an intraoral approach under pentothal sodium anesthesia. A week later, under endotracheal ether anesthesia, the fracture of the right body of the mandible was reduced and immobilized by means of a four-hole stainless-steel bone plate. The approximation was anatomic, as seen in Fig. 11.

For two weeks postoperatively there was seropurulent discharge from the open reduction incision line below the lower border of the mandible. This was not entirely unexpected because of the intraoral compounding, and in view of the open reduction skin incision passing across one of the deep facial lacerations extending into the neck.

The fracture of the left body of the mandible was not treated. Although not in perfect alignment, the position was considered favorable for bony union in an edentulous mandible. (Fig. 12.)

Four and one-half weeks after open reduction, the bone plate was removed. Full upper and lower dentures were constructed and the patient was discharged to duty twelve weeks after injury.



Fig. 11.—Approximation after extraoral open reduction and fixation by means of bone plate and four screws.

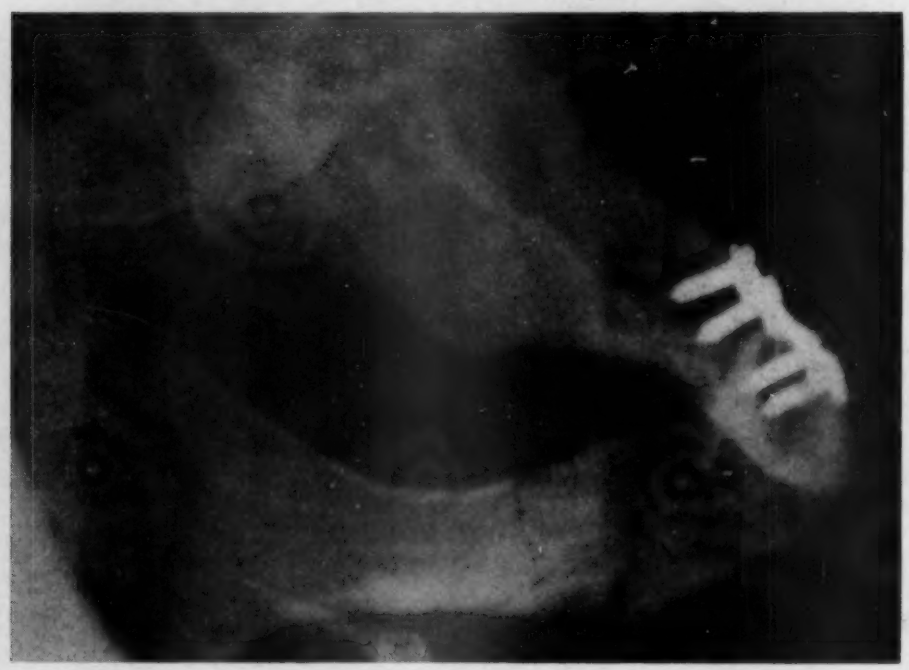


Fig. 12.—Fracture of the left mandible which was not treated.

Comment.—This soldier had multiple injuries and fractures and was severely traumatized. In a series of surgical procedures his fractures were reduced. Under sulfadiazine protection, through a contaminated external approach and in the presence of intraoral communication, a four-hole bone plate anatomically reduced one of the bilateral mandibular fractures. It was agreed that the fracture on the other side was in such alignment as not to warrant surgical intervention. The bone plate was removed at the end of thirty-one days. The functional and anatomic results were good, and in spite of multiple injuries the soldier was restored to full military duty in three months.

BATTLE CASUALTIES

The jaw fractures caused by shell or rifle fire, bombs, grenades, or mines were, with few exceptions, severe injuries in which there was bone loss and varying degrees of soft tissue injury. In addition, associated wounds in the trunk or extremities complicated treatment and prolonged recovery. Infective complications were more common in this group and they required constant and skillful care and nursing in order to minimize deformity and disfigurement. Fewer of these cases were rehabilitated for duty overseas, and the majority were evacuated to the United States for reconstructive surgery.

The etiology of the 104 jaw battle casualties is shown in Table III.

TABLE III

Shrapnel	57	55%
Bullets	29	28%
Mines	18	17%

The following case histories are representative of the three main classifications of jaw fractures derived in combat.

1. Shrapnel.—Case No. 20445. A 21-year-old infantry private was injured in combat by shell fragments on July 11, 1944. He received perforating lacerations of the left cheek, mouth, and left side of the tongue, in addition to a compound, severely comminuted fracture of the left body of the mandible.

The soldier hemorrhaged profusely after being struck and received first aid from a medical corps man. That same day, at the 8th Evacuation Hospital, his cheek and tongue wounds were débrided and multiple shell fragments were removed from the site of injury. The lower right first molar and the lower left posterior teeth, which were shattered and in the lines of fracture, were removed. The jaws were immobilized with Stout continuous loop wiring and elastic traction. Because of massive edema of the floor of the mouth and respiratory difficulties, a tracheotomy was performed and a Levine tube inserted.

Six days after injury he was evacuated to the 6th General Hospital, and shortly after admission he developed a severe secondary hemorrhage from the tongue wound. The elastics were removed and the hemorrhage controlled by means of suturing and pressure. The tracheotomy was still open, the patient had gross edema of the tongue and floor of the mouth and was unable to swallow. (Figs. 13*A* and 13*B*.)



Fig. 13A.—Left lateral jaw showing displacement of large mandibular segment. Note multiple shell fragments embedded in soft tissues.



Fig. 13B.—Anteroposterior view

Twelve days after admission to our hospital, and following sulfadiazine therapy and frequent hot irrigations, direct wiring of the displaced segments through an open reduction was performed under local anesthesia (Fig. 14).

Several small intraoral sequestra were aborted and there was drainage from the open reduction incision for ten days. On September 13, the transosseous wires were removed, and all intermaxillary wiring was removed ten weeks after injury. He was not discharged to duty until three months after injury.

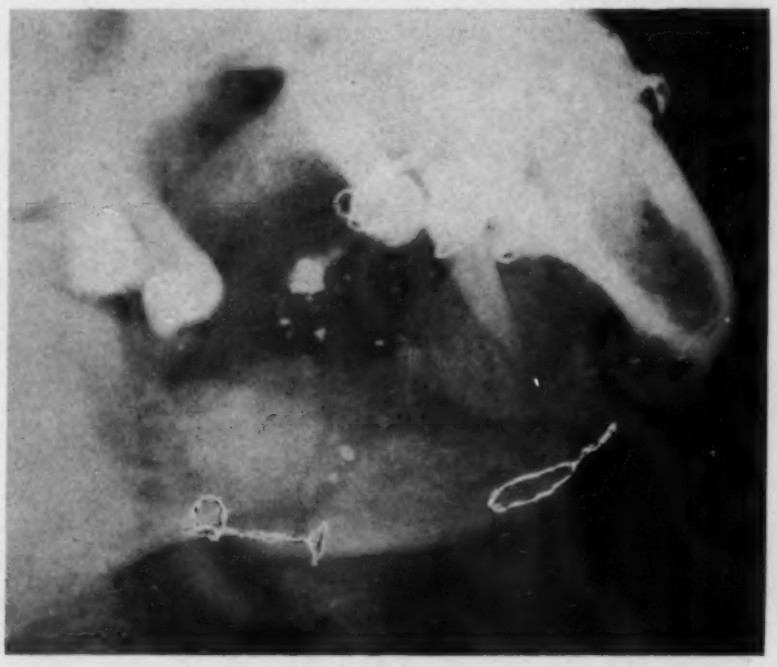


Fig. 14.—Position of fragments after reduction by direct wiring.

Comment.—Facial wounds caused by shell explosion were usually multiple, ragged lacerations, about and communicating with the oral cavity, and the soft tissues were peppered with metallic foreign bodies. The primary closure of these facial wounds, particularly when they were associated with compound jaw fractures, was a radical departure from the accepted technique at the end of World War I and the beginning of World War II. Not all of them remained closed but the majority of them did, and ugly contractures and prolonged hospitalization were thus minimized. Because of some upward displacement of the distal segment, the anatomical result of the above case was graded as *moderate*—the functional result was *good.*

2. Bullets.—Case No. 23448. A 23-year-old infantry sergeant was wounded in action on Sept. 16, 1944, having been struck by sniper bullets in the left cheek and chin, the missiles exiting through the right neck. He received severe comminuted compound fractures of the symphsis and right and left bodies of the mandible. On the day of injury the soft tissue wounds of the cheek, chin, and

neck were débrided and sutured, and the jaws were immobilized by means of continuous loop wiring and elastic traction at a near-by evacuation hospital.

Three days after being wounded, he was admitted to the 6th General Hospital. The day after admission, acute fluctuant swellings in the right and left submental regions were incised and rubber dam drains were inserted.

The lower left first incisor was missing and the apices of the right first incisor and the left second incisor were involved in the comminution. They were very mobile and were removed. There was considerable downward displacement of the comminuted lower border of the mandible—particularly on the right side. A plaster chin sling was applied to exert upward pressure and to help to mold the fragments into better position.



Fig. 15.—Right lateral jaw.

The occlusal relations were good and all inflammatory signs in the right and left submental regions disappeared.

After two weeks the chin sling was removed and more favorable position of the fragments visualized, as seen in Figs. 15 and 16. Nine weeks after injury the elastic traction was removed and there was obvious clinical and functional nonunion in the symphysis region extending over to the left body (Fig. 17).



Fig. 16.—Left lateral jaw.



Fig. 17.—Anteroposterior view. Nine weeks after injury there was clinical nonunion in the midline.

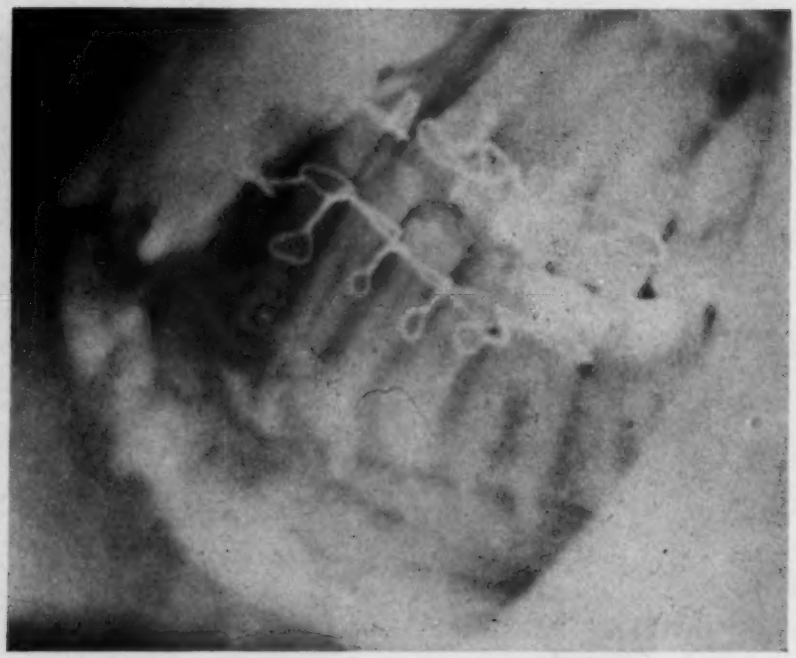


Fig. 18A.—Right lateral jaw which showed considerable bone repair nine weeks after injury.



Fig. 18B.—Left lateral jaw.

X-rays at that time showed favorable bone regeneration in the right body (Figs. 18A and 18B). The lower right second premolar and left canine were extracted and a cast silver splint was cemented to the lower teeth.

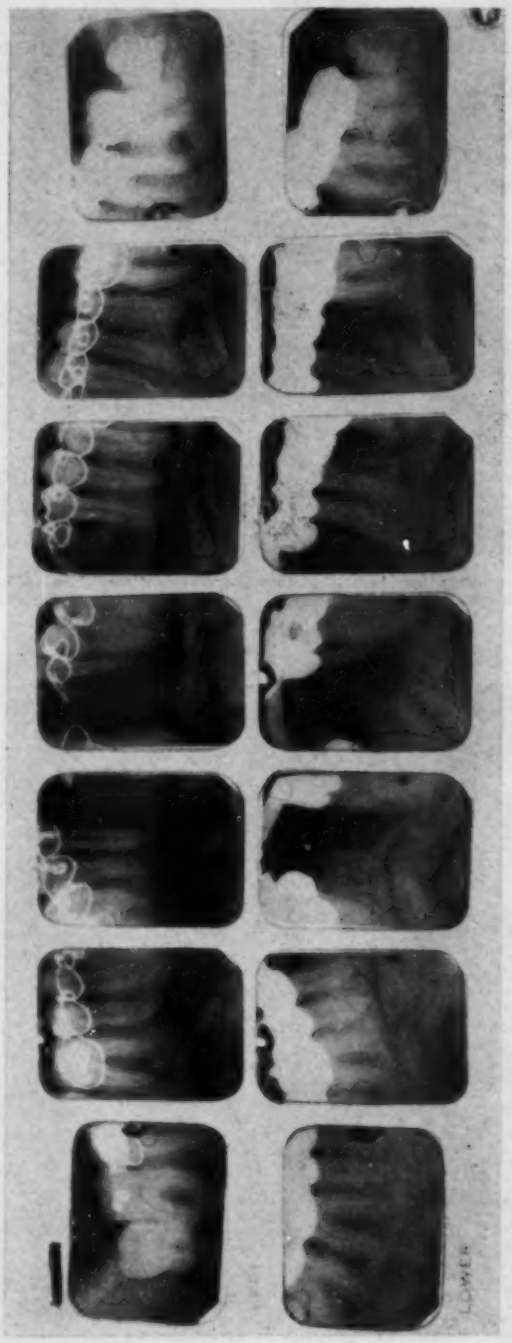


Fig. 19.—The top row of intraoral films are of the lower teeth before bone grafting. The bottom row were taken two weeks after bone grafting (R13 and L11 having been extracted).

On Dec. 1, 1944, cancellous chip bone grafts from the right ilium were packed into the ununited fracture lines in the symphysis and left body after removing the interposed fibrous tissue.

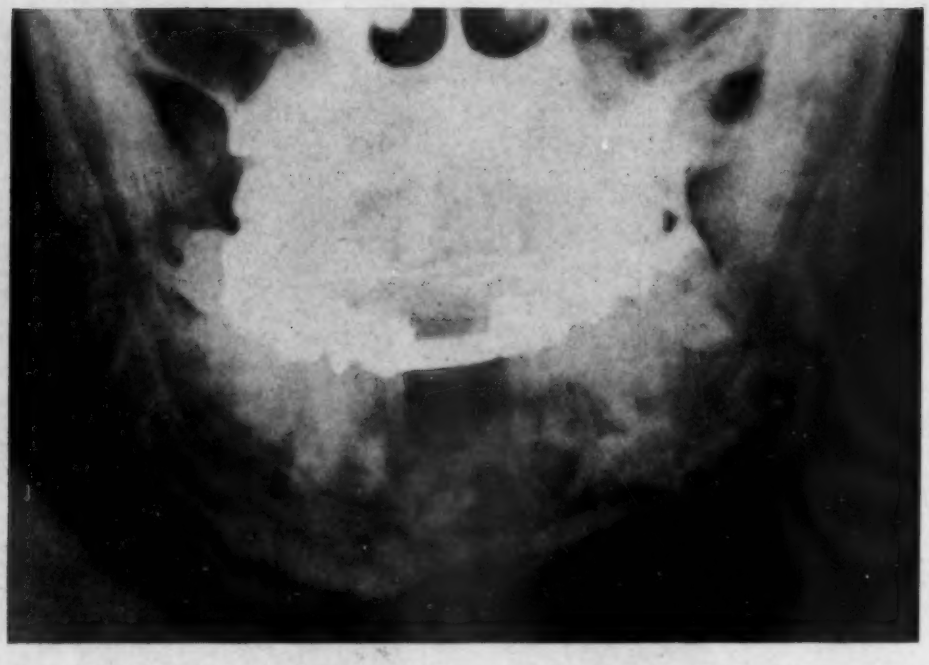


Fig. 20.—Anteroposterior view two weeks after cancellous bone chips were packed into the symphysis and left side fractures. The lower jaw is immobilized with a cast metal cap splint.

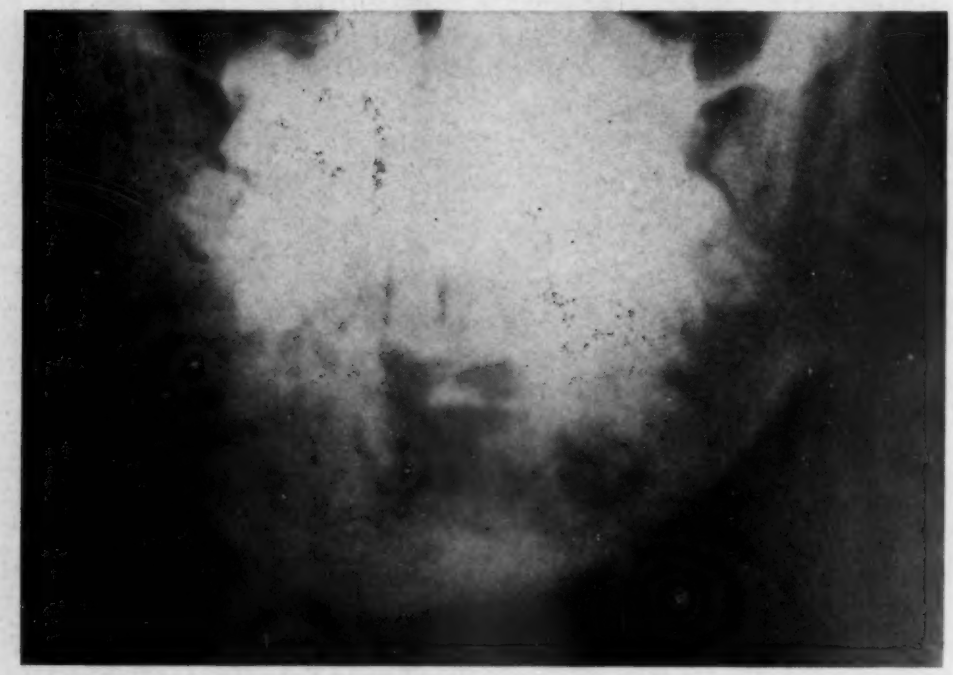


Fig. 21.—Anteroposterior view ten weeks after bone grafting. There were firm bony union and good occlusion.

It is of interest that at the time of bone grafting an accidental perforation of the oral mucosa was made, establishing a communication between the mouth



Fig. 22.—Right lateral jaw.



Fig. 23.—Left lateral jaw.

and the site of bone grafting. The patient was given parenteral penicillin; the chart remained flat; there were no signs of sepsis, and very satisfactory union of the bone chips resulted. (Figs. 19 and 20.)

Eight weeks after bone grafting the cast splints were removed. There was firm bony union and the occlusion was undisturbed. A lower partial denture was inserted, and the patient was discharged to duty on March 3, 1945. (Figs. 21, 22, and 23.)

The continuity of the lower border was established and the architecture of the mandible was restored. Compare with Figs. 15, 16*A*, and 16*B*, at the time of injury.

Comment.—This case represents very severe comminuted fractures of the mandible in which conservative treatment maintained occlusal and teeth relationships, but there was sufficient loss of bone to result in fibrous union. Cancellous chip bone grafts from the iliac crest, in spite of oral contamination, resulted in rapid bony union and restored normal architecture.

3. Mines.—Case No. 20673. A 22-year-old private was injured by a mine explosion while on reconnaissance patrol. The soldier directly in front had stepped on a German mine and was instantly killed. The patient received multiple penetrating wounds of the face, neck, shoulders, and hands. The wounds were débrided and numerous metallic foreign bodies were removed.

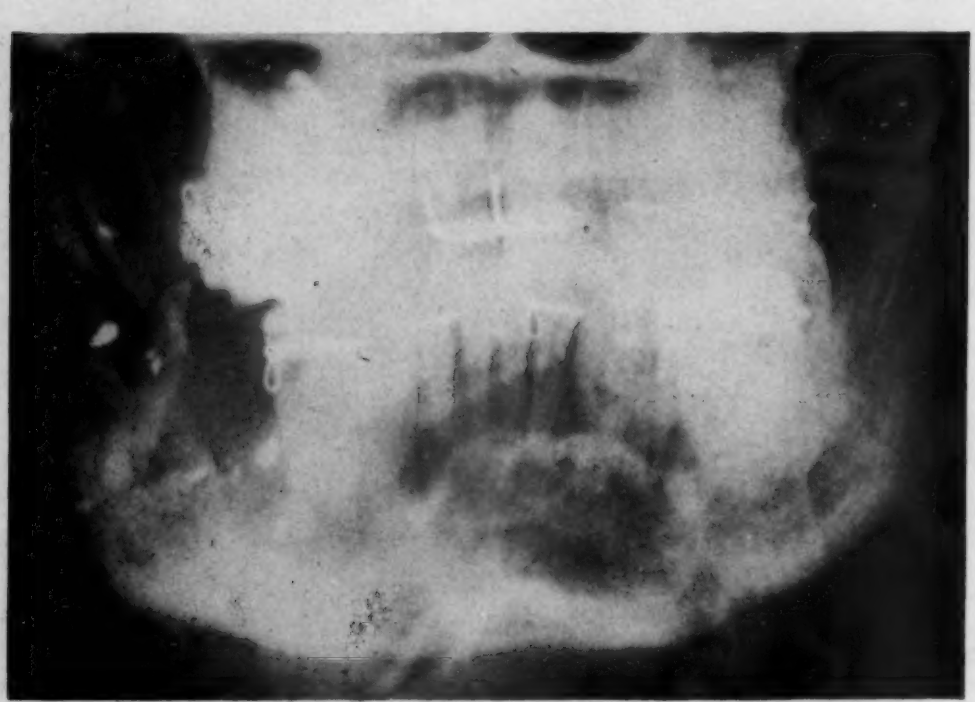


Fig. 24.—Anteroposterior view showing crushing effect the mine explosion had on the ramus, angle, and body of the left mandible.

On admission to our hospital two months after injury, the jaws were immobilized with continuous loop wiring. The left angle and ascending ramus of the mandible were badly comminuted, and scattered fragments of the mine were visible by x-ray. (Figs. 24 and 25.)

There was continued suppuration about the facial wounds, there was a peripheral left facial paralysis and a ruptured left eardrum. He was evacuated to the United States six days after admission for further hospitalization and treatment.

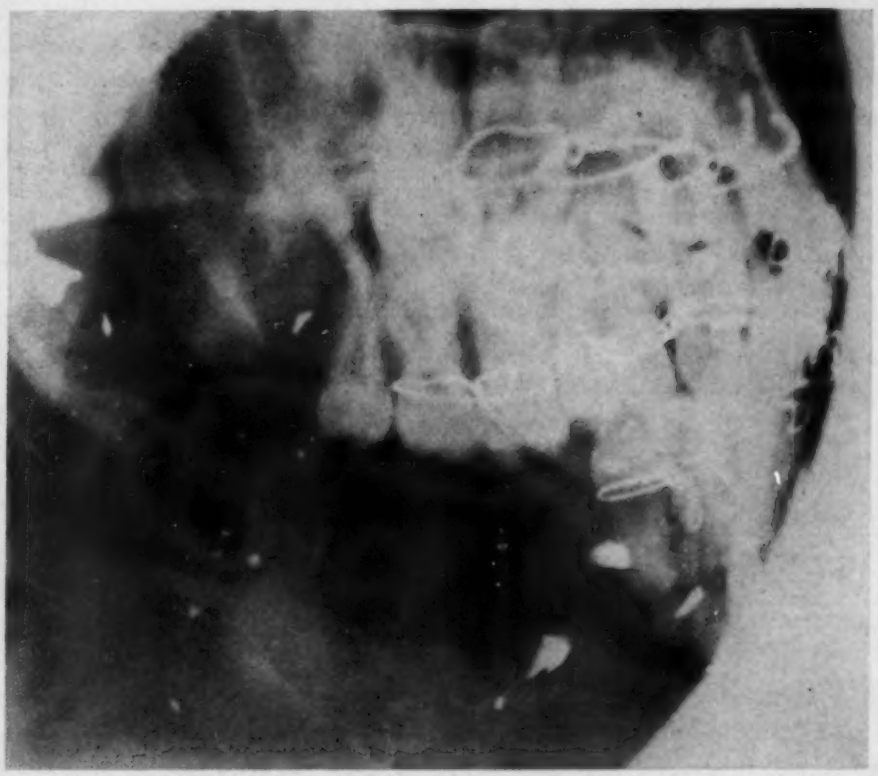


Fig. 25.—Left lateral jaw, showing comminution and bone loss as a result of mine explosion. Several small metallic fragments of the mine are visible.

Comment.—Some of the most devastating facial wounds were caused by mines. The explosion drove dirt, wood, fragments of the mines, and other foreign bodies into the face, neck, hands, and even the trunk and covered parts of the extremities. Ruptured eardrums, loss of vision in one or both eyes, and facial paralysis were frequent sequelae. The jaw fractures were usually severe ones and the pigmented, ragged, contaminated soft tissue defects among the most disfiguring. Rehabilitation overseas in this group was infrequent.

TEETH IN THE LINE OF FRACTURE

In this group of 200 cases, 72 patients had teeth in the fracture lines. At the beginning of the war we adhered to a conservative policy in regard to the controversial subject of when teeth in the line of fracture should be removed. Whenever a tooth was considered important either to reduce the fracture or to maintain the segments in good alignment (even though the apex was involved in the fracture), the tooth was temporarily retained. If the tooth in

question was not essential for reduction and alignment, prophylactic removal was carried out. The incidence of continued suppuration and delayed union and a few cases of nonunion were attributed directly to teeth left in the fracture sites.

A fixed policy was then established that *any* and *all* teeth whose apices were included in the path of the fracture should be removed regardless of their strategic location or importance. Any tooth whose vitality was destroyed was considered a potential source of infection and a definite deterrent to normal bone repair. It was felt that under no circumstances was the temporary retention of potential sources of sepsis justified even though chemotherapeutic agents such as the sulfonamides and penicillin suppressed frank signs of infection. The number of methods available to treat all varieties of jaw fracture made the retention of any involved teeth unnecessary.

LOCATION OF THE FRACTURES

Two hundred seventy-eight fractures of the jaws (among the 200 patients reported) were tabulated as follows:

TABLE IV

LOCATION	NUMBER FRACTURES	RIGHT	LEFT
Symphysis	37		
Premolar and Molar	95	49	46
Angle	72	34	38
Coronoid	5	2	3
Condyle	35	17	18
Maxilla	34		

SYMPHYSIS FRACTURES

In our experience, fractures of the mandibular symphysis, even when uncomplicated by severe comminution or bone loss, were the most serious and difficult of the jaw fractures. The mechanics of muscular contraction pulling the fractured halves of the mandible toward the midline, the ease with which symphysis fractures were displaced, and the longer period of immobilization and fixation required before bony union was established, led to these conclusions:

1. *Symphysis fractures warrant the most rigid fixation and complete immobilization.* The British method of open, cast metal splints or labial arch bars soldered to copper bands which bridged the fracture, plus intermaxillary fixation, worked well. It was essential to obtain an impacted approximation of the two segments and hold that position rigidly. The routine methods of eyelet, loop, intermaxillary wiring, or intermaxillary elastic ligation were found to be inadequate to resist counter muscle pull in these midline mandibular fractures.

2. *Symphysis fractures were slow to unite* and required eight to ten weeks and in some cases twelve weeks of fixation before mobility could not be demonstrated in normal jaw movements.

3. *Symphysis fractures with severe comminution or bone loss* that are likely candidates for nonunion are best treated by radical débridement of the loose,

detached bony spicules, leaving no possible foci for development of a fistula. After clean healing is established, cancellous chip bone grafts from the ilium restore union and full function in from six to eight weeks after operation.

FRACTURES IN THE PREMOLAR AND MOLAR REGION

The dentition of the average American soldier was sufficiently complete so that various methods of intraoral or interdental wiring gave uniformly good results. Stout's technique of continuous loop wiring with intermaxillary elastics was the most universal method employed in the 95 fractures in the molar and premolar regions. Arch wires ligated to individual teeth and fixed to the opposing jaw by means of tie wires or elastic bands were popular techniques, particularly in bilateral fractures of the mandible.

Intermaxillary fixation by direct wiring was not employed prior to evacuation by ship or plane where there was a possibility of sickness. Instead, elastic traction was preferable.

FRACTURES AT THE ANGLE

When an erupted third molar (whose apices were uninvolved in the line of fracture) was present in the distal segment and able to be occluded with its antagonist above, the reduction of angle fractures offered no special problem. When a posterior edentulous fragment was displaced upward and medially, open reduction and direct interosseous wiring was our method of choice. By this method of direct wiring, anatomic alignment was obtained and there were no unfavorable postoperative sequelae. We preferred open reduction and direct wiring to the external skeletal fixation technique.

In describing and classifying fractures at the angle, we adopted the descriptive method of Fry, Shepherd, McLeod, and Parfitt.* Briefly, this was a method of terming angle fractures as *"horizontal favorable"* (H.F.) if the direction of the fracture line was such that it resisted the action of the elevator muscles in pulling the ramus segment upward (Figs. 26 and 27). When the direction of the fracture line was reversed, i.e., as in Figs. 28 and 29 and the posterior fragment was elevated by the temporal muscle, the fracture was termed *"horizontal unfavorable"* (H.U.).

As the pull of the internal pterygoid is stronger than the action of the masseter, many of these angle fractures were displaced lingually or medially. When the direction of the fracture line allowed lingual displacement of the posterior fragment, the fracture was listed as *"vertical unfavorable"* (V.U.) when viewed from above as in Figs. 30*A* and 30*B*.

A line of fracture as in Figs. 31*A* and 31*B* would mechanically prevent the internal pterygoid from displacing the ramus medially so the fracture was classed as *"vertical favorable"* (V.F.). When the angle fracture was such that it allowed upward displacement of the ramus, but no medial or lingual displacement, it was termed *"horizontal unfavorable," "vertical favorable"* (H.U.—V.F.) (Fig. 32).

*The Dental Treatment of Maxillo-facial Injuries, Oxford, 1942, Blackwell Scientific Publications Ltd.

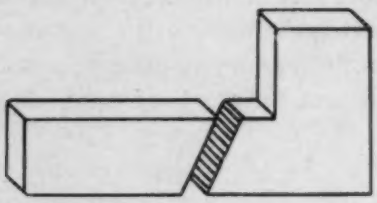


Fig. 26.

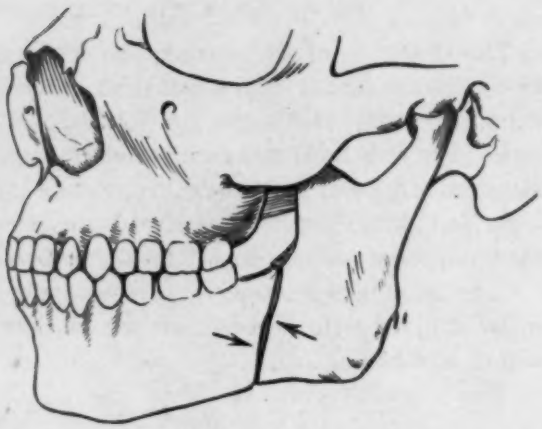


Fig. 27.

Fig. 26.—The direction of the fracture line is such that upward displacement of the distal segment is prevented by the inclined plane above horizontal favorable (H.F.).

Fig. 27.—Horizontal favorable (H.F.) fracture at the angle with little if any upward displacement of the ramus.

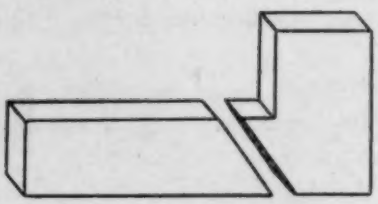


Fig. 28.

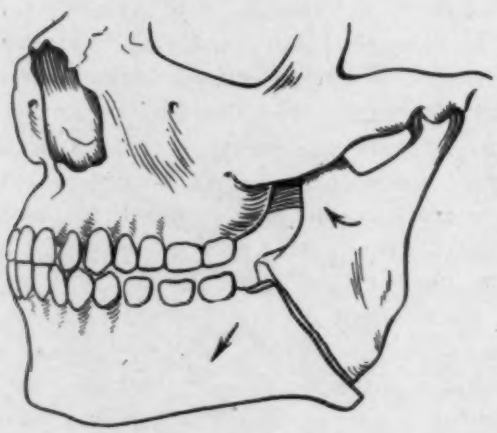


Fig. 29.

Fig. 28.—When the direction of the fracture line is as above, the ramus fragment is displaced upward. Such a fracture was termed "horizontal unfavorable" (H.U.).

Fig. 29.—Horizontal unfavorable (H.U.) fracture at the angle.

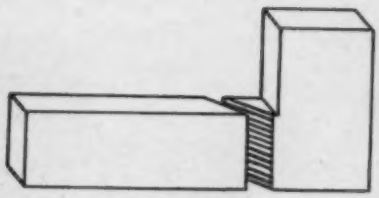


Fig. 30*A*.

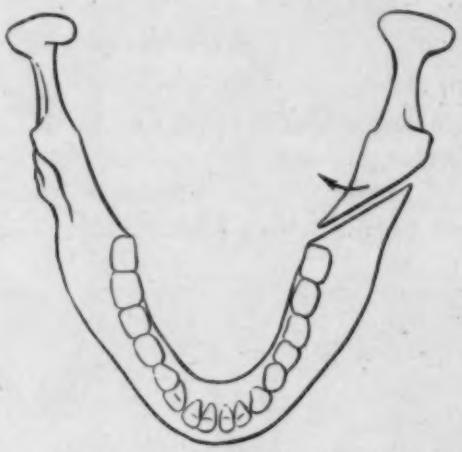


Fig. 30*B*.

Fig. 30*A*.—When the direction of the fracture allowed the internal pterygoid muscle to pull the ramus segment lingually, the fracture was termed "vertical unfavorable" (V.U.).
Fig. 30*B*.—Vertically unfavorable (V.U.).

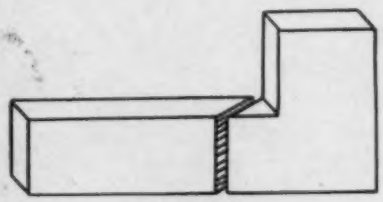


Fig. 31*A*.

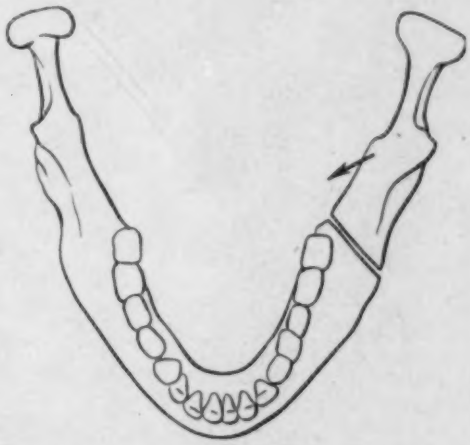


Fig. 31*B*.

Fig. 31*A*.—In this diagram the pull of the internal pterygoid is not able to displace the distal segment because of the direction of the fracture line. Vertically favorable (V.F.).
Fig. 31*B*.—Vertically favorable (V.F.).

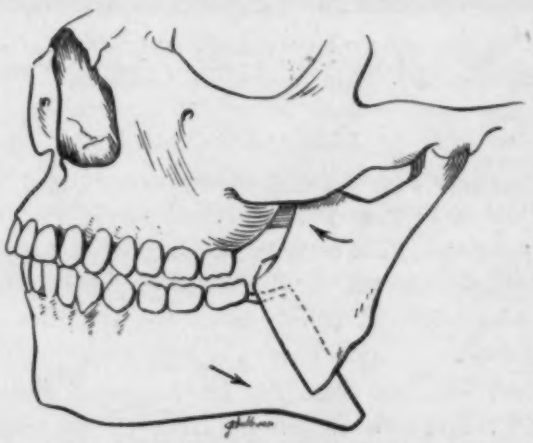


Fig. 32.—Horizontal unfavorable, vertical favorable fracture (H.U.—V.F.).

FRACTURES OF THE CORONOID PROCESS

In this series there were five fractures of the coronoid process—two the result of accidental injuries and three the result of enemy fire. Those caused by machine gun or shell fragments were markedly comminuted and characterized by severe trismus due to cicatricial bands in the temporal muscle attachments. Parotid fistulas were secondary complications in two of these cases.



Fig. 33.—Fracture dislocation of the left condyle. There is also a subcondylar fracture on the right side plus a vertical fracture of the right ramus. At the lower border in the region of the left canine there is a displaced triangular segment.

FRACTURES OF THE CONDYLE

All condylar fractures were treated conservatively and no open reductions were performed either to replace the condyle head in the fossa or to align it with its ramus attachment. There were 35 fractured condyles confirmed by x-ray, and it is very likely that an equal number escaped detection. Five of the patients tabulated had *bilateral* fractures of the condyles with displacement *from the articular fossae.*

The soldier whose bilateral condylar fractures are shown in Fig. 33 was injured when a truck overturned. In addition to the condyle fracture dis-

locations, he had a compound fracture at the left canine region with a displaced triangular segment at the lower border. Twelve weeks after injury he was discharged to military duty with full functional use of his jaws, no trismus or other complications.

Table V shows the disposition and functional results of the 30 patients (35 condyle fractures). In 3 patients trismus was so severe and the jaw function so restricted that evacuation to the United States was carried out. The other 7 patients were evacuated for multiple wounds and for other than jaw complications—such as loss of eye, ruptured eardrum, facial paralysis, etc.

TABLE V

DISPOSITION		FUNCTIONAL RESULT		
DUTY	EVACUATED TO U. S.	GOOD	MODERATE	BAD
20	10	22	5	3

Several of these cases demonstrated that efficient jaw function was not dependent on perfect positioning of the head or neck of the condylar process. The fibrous joints resulting from displacement, with few exceptions, allowed efficient mastication and no unfavorable sequelae at the time of disposition.

MAXILLARY FRACTURES

The most variable combinations of fractures of the maxilla were represented in the 34 patients so afflicted. The gunshot wounds invariably shattered the lateral maxillary walls and created oroantral fistulas. Although severe, these gunshot wounds of the maxilla rarely displaced the upper jaw from its other cranial attachments. The problem was largely one of débridement, comminution, and bone loss, in contrast to the problem of moving the entire maxillary structure upward and forward as in the depressed fractures associated with jeep accidents.

CONCLUSIONS

The sound and tested techniques on the treatment of military jaw casualties passed down from experiences gained in World War I were valuable inheritances to meet the maxillofacial problems of World War II. Time-consuming appliances, complicated, elaborate apparatus were not employed. Simplicity, by the various closed methods of intraoral wiring, in most cases fulfilled all the requirements of good fracture treatment. In others, open reduction and direct coaptation of displaced jaw fragments by transosseous wiring or bone plating gave excellent results without postoperative bone infections. To restore jaw architecture following bone loss, and in cases of nonunion, cancellous chip bone grafts from the ilium was a gratifying procedure. Under chemotherapy protection two of the cases of cancellous bone grafting responded uneventfully even though complicated by communication with and contamination from the oral cavity.

Severe sepsis, respiratory complications, and the hideous deformities associated with war wounds of the jaws were exceptions, and in large measure their absence was related to the primary closure of the soft tissue wounds.

TABLE VI

Of the 200 patients with jaw fractures:
130 (65%) were discharged to duty
70 (35%) were evacuated from our hospital to the United States for further hospitalization and treatment

TABLE VII

	GOOD	MODERATE	BAD
Anatomical result	139	43	18
Functional result	143	42	15

At the time of disposition the anatomical and functional results were evaluated as shown in Table VII.

The treatment of these 200 jaw fractures in the 6th General Hospital was the combined responsibility and represented the cooperative efforts of a plastic surgeon and an oral surgeon working together as a maxillofacial team.

CASE REPORTS FROM THE MAXILLOFACIAL TEAM, 6TH GENERAL HOSPITAL

MAJOR SOMERS H. STURGIS, M.C., AND MAJOR DANIEL J. HOLLAND, JR., D.C.

Surgical Correction of Mandibular Prognathism in Two Edentulous Patients

SEVERE mandibular protrusion with associated extreme malocclusion was observed infrequently among our overseas troops. No doubt, detection and screening out of the marked cases by draft board preinduction examination and subsequent rejection or deferment reduced the number of such anomalies in the military service. Inability to chew the Army ration was a disqualifying defect and legal incapacity for active military duty.

The presence of natural teeth are usually important adjuncts in the surgical correction of prognathism. They not only serve as guides for estimating the amount of reduction in the size of the mandible, but as sources of fixation and retention postoperatively. The maintenance of the midline jaw relationships and vertical dimensions are also facilitated when some of the natural dentition remain.

Increased masticatory function and improvement of facial deformity may occasionally be effected by the extraction of all remaining teeth and the construction of complete dentures—without further surgical intervention. The prostheses when carefully equilibrated restore functional teeth relationships and improve the characteristic profile of mandibular protrusion.

Two patients were admitted to the 6th General Hospital in Rome who were endentulous and whose mandibles were so markedly protracted that functional prostheses were impossible because of the extreme discrepancy between the maxillas and mandibles.

CASE NO. 25763.—A 30-year-old sergeant was admitted to the 6th General Hospital on November 2, 1944, with a diagnosis of gastritis and marked mandibular prognathism (Fig. 34). In spite of a severe Class III malocclusion and difficulty in eating, he had been inducted into the Army and shipped overseas. Unable to chew the Army diet, he developed upper abdominal distress and stomach cramps.

At a previous military hospital his remaining upper and lower teeth had been extracted, and it was planned to construct dentures to provide a normal degree of masticatory efficiency with an aim to eliminate his digestive disturbances (Fig. 35).

Because of the unfavorable ridge relationships (Fig. 36) it was felt that dentures would neither be stable nor comfortable, and surgical correction to reduce the mandibular protraction was elected. (Figs. 37, 38, 39, and 40.)



Fig. 34.—Photograph of patient with mandibular prognathism.



Fig. 35.—Right lateral jaw x-ray before teeth were extracted showing relationship between mandibular and maxillary arches.

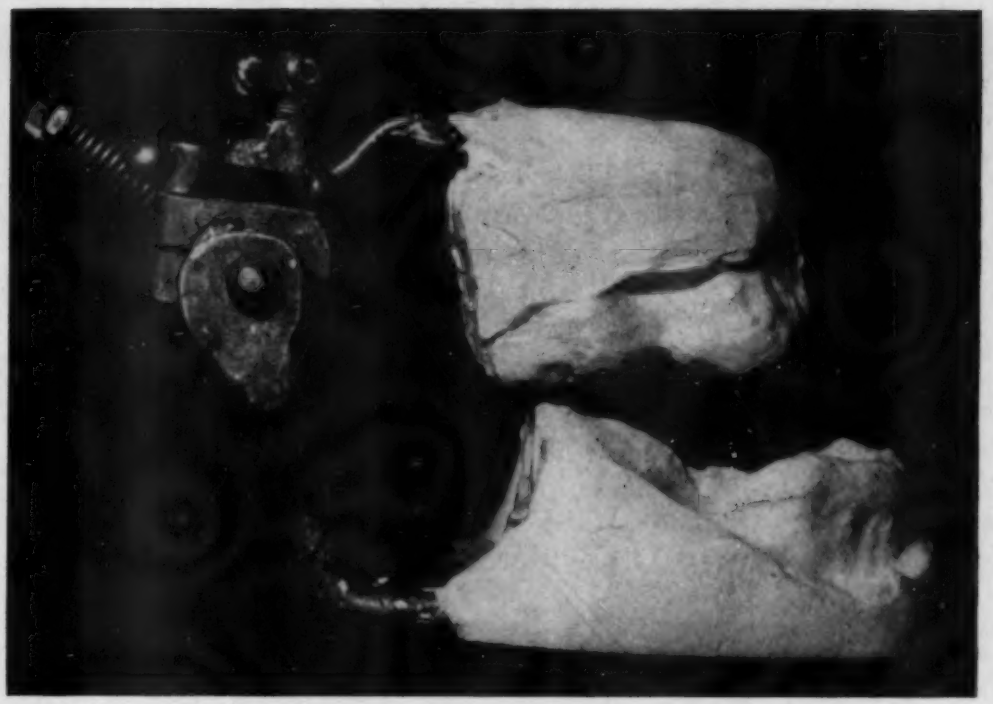


Fig. 36.—Articulated models, showing protraction of mandible and discrepancy between anterior ridge of maxilla and mandible.

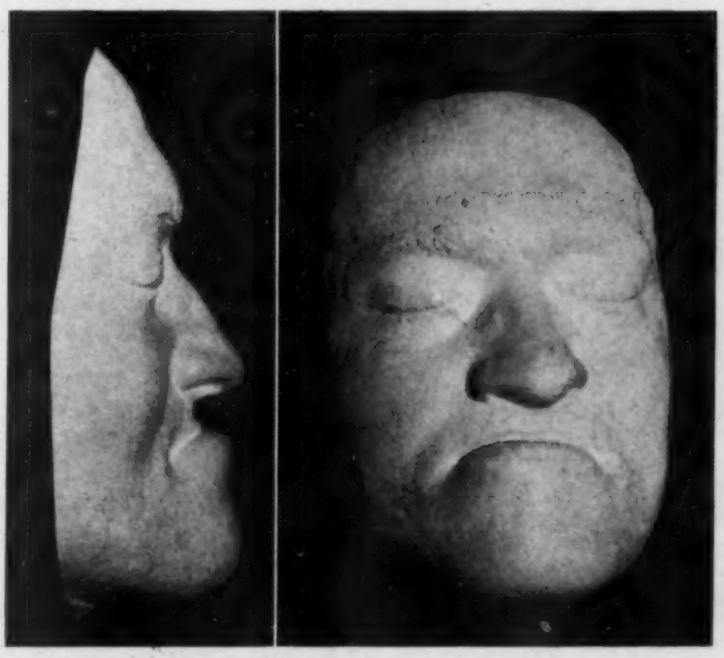


Fig. 37. Fig. 38.

Fig. 37.—Lateral view of face mask before operation.
Fig. 38.—Full face view of face mask.



Fig. 39.—Preoperative profile x-ray.



Fig. 40.—Right lateral jaw showing length of horizontal rami.

Operation.—Under endotracheal ether anesthesia 5 cm. incisions were made slightly below the lower borders of the bodies of the right and left mandibles. By combination Gigli saw and dental drills, 20 mm. segments of mandible were resected bilaterally without entering the oral cavity. The chin was set back and the separated anterior and posterior sections were coaptated and fixed by means of four-hole Sherman bone plates, as shown in Figs. 41, 42, and 43.

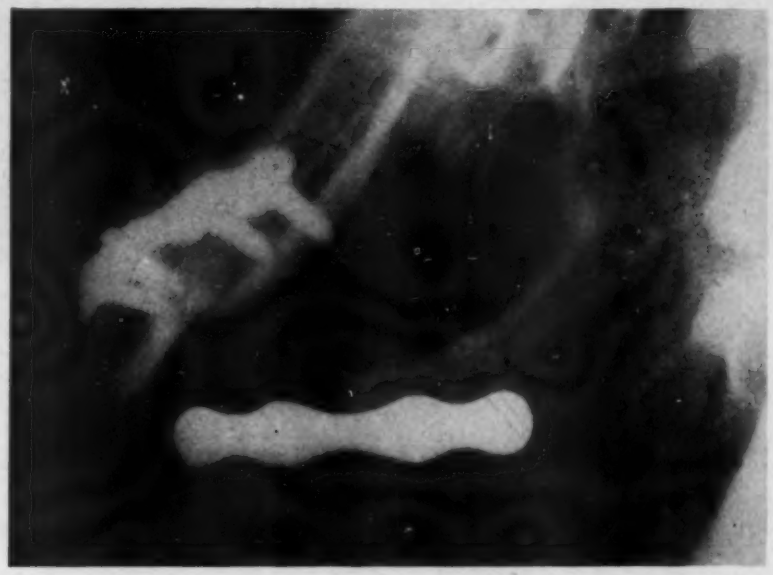


Fig. 41.—Right lateral jaw plate after resection of 20 mm. blocks of mandible bilaterally—four-screw Sherman bone plates were used to coaptate and fix the separated segments.



Fig. 42.—Left lateral jaw. Cancellous bone chips from the resected blocks were packed around the fracture line under the bone plate.

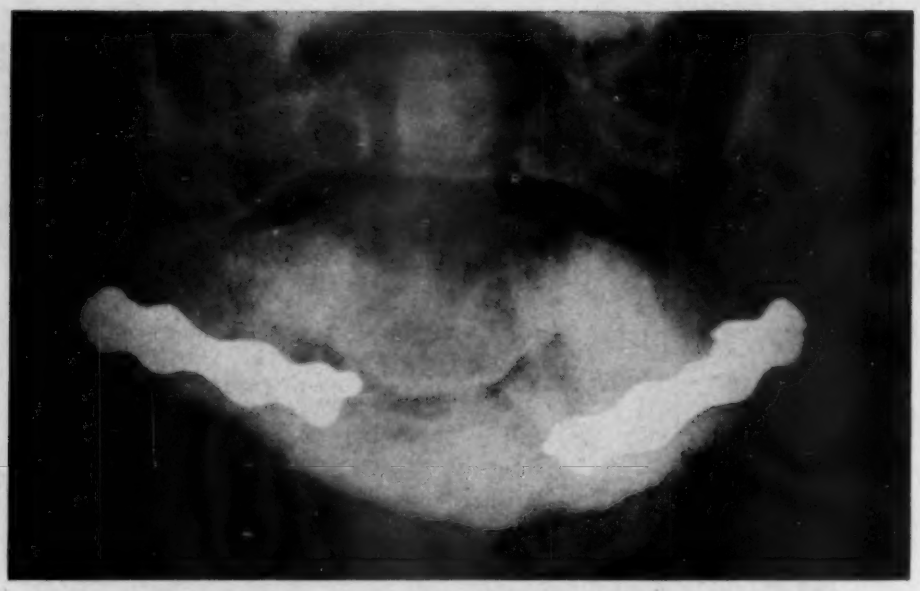


Fig. 43.—Anteroposterior view showing chin set back and held by bone plates.

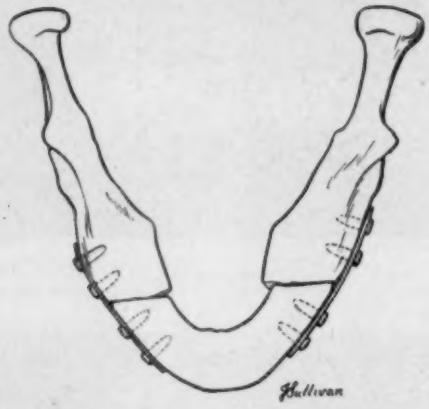


Fig. 44.—Diagram illustrating the difference in width of the anterior and posterior segments. The lingual edges of the distal segments caused prominent intraoral projections.



Fig. 45.—Front view one month postoperatively. There was slight "double chin" as a result of retraction of the mandible.

Fig. 46.—Lateral view. Compare with Fig. 34.

The skin sutures were removed on the sixth postoperative day and the recovery was uneventful. There was anesthesia of the lower lip and prominent intraoral bony projections of the distal segments lingually—caused by the difference in the width of the anterior and posterior segments (Fig. 44). Full upper



A. *B.*

Fig. 47.—*A*, Preoperative profile face mask. *B*, Postoperative profile face mask.



A. *B.*

Fig. 48.—*A*, Preoperative mask. *B*, Postoperative mask.



Fig. 49.—Lateral jaw eleven weeks postoperatively. The bone plates have been removed. There was solid union and patient was comfortably wearing upper and lower dentures.



Fig. 50.—Anteroposterior x-ray showing union bilaterally, dentures inserted.

and lower dentures were inserted one month after operation (Figs. 45, 46, 47, and 48).

On Jan. 25, 1945 (ten weeks postoperatively), the bone plates were removed. The fractures were firmly united. The patient was discharged to full military duty wth complete relief of his original epigastric complaints. (Figs. 49 and 50.)

One and one-half years after operation the hypesthesia of the lower lip had disappeared and full sensory and functional activity of the jaws and associated tissues was present.



Fig. 51.—Sketch to represent prognathic mandible and the relations of the edentulous jaws.



Fig. 52.—Preoperative profile face mask.

Case No. 25390.—A 24-year-old soldier was transferred from a near-by hospital to the 6th General Hospital on Oct. 31, 1944. At the previous hospital all his remaining teeth were extracted because of multiple periapical abscesses and cervical adenopathy. There was pronounced overdevelopment of the mandible and the ridge relationships were as diagrammed in Fig. 51. (Figs. 52 and 53.)

Operation.—Bilateral partial jaw resection was performed to remove 20 mm. blocks of bone from the right and left bodies of the mandible under endotracheal ether anesthesia (Fig 54). Six-centimeter incisions were made at the right and left lower borders of the mandible from the angles forward; the external maxillary arteries were retracted, and the periosteum was stripped from the segments to be removed. By means of Gigli saw, dental drills, and osteotomes the



Fig. 53.—Preoperative right lateral jaw which shows long horizontal rami and prominent symphysis.

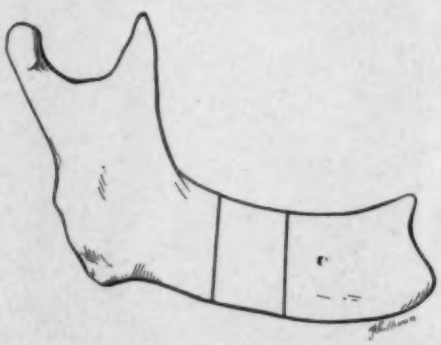


Fig. 54.—Diagram showing location of 20 mm. sections of mandible removed from right and left sides.

lingual and buccal bone cortices were cut through and the measured blocks of bone fractured free (Fig. 55). The mandibular arteries were grasped and tied and the chin set back (Fig. 56) to approximate the separated bone ends held by Sherman four-hole bone plates. Cancellous bone chips from the resected blocks were packed around the fracture lines and under the Sherman plates (see Figs. 41 and 42).



Fig. 55.—Diagram illustrating removal of block of mandible by means of Gigli saw. The posterior section has been completed and the mandibular artery tied.

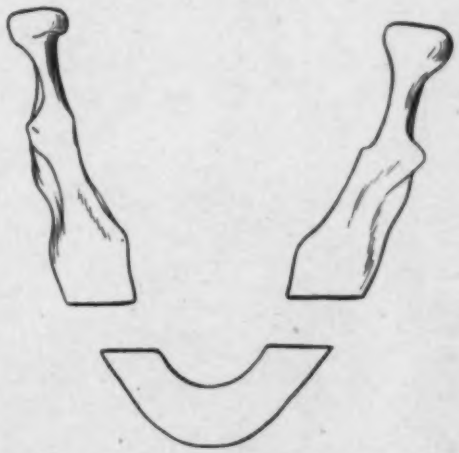


Fig. 56.—Illustration to show the distance chin was set back following resection.

A prominent "double chin" and submental fullness resulting when the chin was repositioned (Fig. 57) was excised by connecting the right and left incisions and removing a 22 mm. strip of excess soft tissue. The wound was closed in layers and the skin edges were carefully approximated with silk sutures. A 500 c.c. blood transfusion was administered immediately after the operation, and, with the exception of a hematoma on the right side, the recovery was normal. The skin sutures were removed on the seventh postoperative day. Two days



Fig. 57.—Note the "double chin" after the chin was repositioned. By connecting the right and left incisions and removing 22 mm. strip of excess skin, this fullness was corrected.



Fig. 58.—Lateral jaw six weeks after resection and immediately following removal of bone plates. There was firm union on both sides.

later the patient was transferred to a military hospital in Naples where the bone plates were removed and maxillary and mandibular dentures constructed. He was discharged to full military duty three months after the operation. (Figs. 58 and 59.)



Fig. 59.—Anteroposterior view following removal of bone plates.



Fig. 60. Fig. 61.

Fig. 60.—Photograph of patient one and one-half years after surgical correction of prognathism. These are the original dentures inserted ten weeks after operation.

Fig. 61.—Profile. Compare with preoperative mask, Fig. 52.

Figs. 60 and 61 were taken one and one-half years after operation. The original dentures were still being worn and patient had full function.

Without surgical correction these two cases of marked mandibular prognathism would have been evacuated from active theaters of war at a time when

man power and replacements were critical. They were restored to military duty with greatly improved masticating efficiency and improved facial configuration. Bone plating proved a satisfactory method of fixation in both cases.

Case Report of Adenoadamantoblastoma

A 17-year-old male British subject was admitted (by special permission) to the 6th General Hospital while stationed in Casablanca, French Morocco, in February, 1944.

Chief Complaint.—Painless swelling at the angle of the left mandible and a gradually increasing intraoral mass in the lower left molar region.

History.—Six years before admission (1938) it was noticed that the mandibular left second molar had not erupted, while its opposite on the right side was in normal occlusion. Dental radiographs revealed a compartmented follicular cyst associated with the crown of the unerupted second molar, and examination disclosed some bulging of the buccal alveolar plate in that region (Figs. 62 and 63).

In Paris, on July 15, 1938, under general anesthesia the cyst sac was enucleated and dissected from the crown of the involved molar—without disturbing the tooth. A microscopic, pathologic report of follicular cyst was made, and the recovery was uneventful. Within a year the second molar began to erupt.

Two years before admission (1942) the patient noticed a slight swelling in the buccal sulcus of the lower left molar area and some prominence at the angle of the left mandible. X-rays were taken and reported as negative, and the patient's parents were reassured. The intraoral mass slowly increased in size and the facial asymmetry became more pronounced.

Physical Examination.—A firm, nontender, nonfluctuant mass was palpated anterior to the angle of the left mandible. Intraorally there were marked bulging of the buccal and lingual alveolar plates, parchmentlike crepitus, no fistula. X-rays were as shown in Figs. 64 and 65.

A diagnosis of adamantoblastoma was made and the operation was planned in two stages:

1. *Intraoral.*—Under local anesthesia the second premolar, first molar, second molar, and unerupted third molar were extracted (Figs. 66 and 67). Cystic tissue adherent to the bifurcation of the first molar, plus a 4 by 4 mm. biopsy specimen from the cystic lesion were removed for microscopic examination. The gingival edges at the sites of the extractions were approximated with silk sutures to hasten closure of the intraoral communication.

The biopsy report revealed cysts of varying size lined by tall columnar ameloblasts which in some fields were laying down normal enamel and in other fields a mucoid material which filled some of the cysts.

Diagnosis: Adenoadamantoblastoma.

2. *Extraoral.*—Five weeks after the extractions, when the oral communication with the cyst cavity through the sockets had healed, the second stage of the operation was performed. This interval was deemed advisable because in the event of a complete resection of the involved mandible a bone graft was contemplated, and, therefore, absence of oral contamination was essential.



Fig. 62.—Left lateral jaw radiograph taken in July, 1938, disclosing a large follicular cyst associated with the crown of the unerupted left second molar. Patient was 11 years old.



Fig. 63.—Right lateral view showing eruption and normal positioning of the right second molar tooth.



Fig. 64.—Left lateral jaw (February, 1944) showing honeycombed, multilocular cystic area in the first molar region. The second molar, shown in Fig. 62, had erupted.



Fig. 65.—Occlusal view of left molar region showing bulging of both buccal and lingual alveolar plates.



Fig. 66.—Left lateral jaw after removal of the second premolar and the molar teeth. Biopsy was taken at this time.



Fig. 67.—Occlusal view following extractions.

An 8 cm. incision was made just below the inferior border of the left mandible, the external maxillary artery was ligated, and the entire body of the mandible from the angle anteriorly was uncovered, exposing the tumor mass. The cystic, honeycombed mass and a generous border of normal bone were excised and curetted, and the excised borders were coagulated with endothermy.

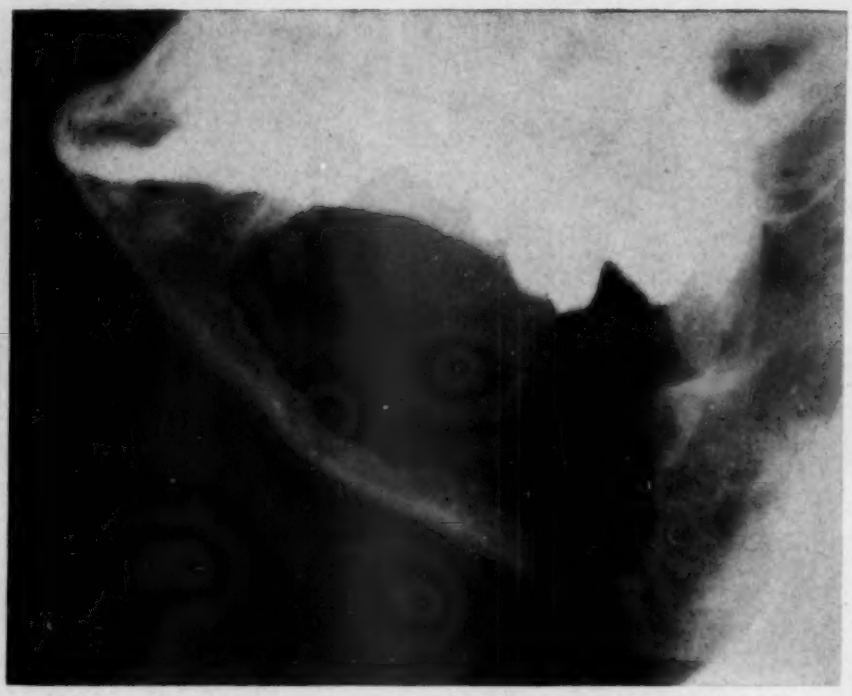


Fig. 68.—X-ray appearance of left mandible following radical excision of tumor mass.

All that remained was the periosteum of the lower border and a thin strip of bone (Fig. 68). The wound was closed in layers and the skin edges carefully approximated with multiple fine interrupted sutures. Pressure bandage dressing was applied and the postoperative convalescence was smooth. Intermaxillary wires were applied to immobilize the mandible, and the patient was cautioned about the possibilities of fracture at the operation site. The skin sutures were removed on the fifth and seventh days.

The gross specimen was described as a 3 by 2.5 cm. mass of honeycomb-like cancellous bone plus a number of smaller fragments. Contained within these shell-like, cystic compartments were masses of soft, meaty-red tissue. The microscopic analysis showed variable structure, but the essential feature consisted of acini of dark blue-staining epithelial cells which were cuboid, and occasionally columnar ameloblasts. The acini enclosed a matrix of fibrous tissue and in the smaller buds was a glairy amorphous secretion. Superimposed was an acute and chronic inflammation indicated by bulging capillaries, proliferating fibroblasts, and large numbers of diffusely spread inflammatory cells, chiefly neutrophiles.

Diagnosis: Adenoadamantoblastoma.

Figs. 69 to 72 demonstrate the bony regeneration following operation. A lower partial plate was constructed to prevent further elongation of the maxillary teeth and the patient was symptom free two years after operation.

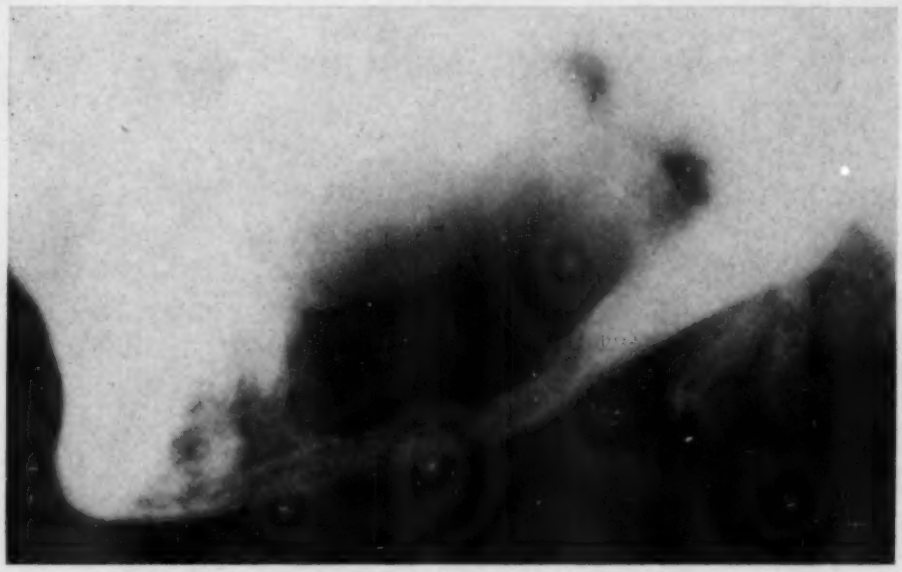


Fig. 69.—X-ray ten weeks postoperatively. There is rapid regeneration of bone at the lower border and anterior edge.



Fig. 70.—Eight months postoperatively.

Comments.—This case may well represent an adenoadamantoblastoma derived from the epithelium of the original follicular cyst (Fig. 62) operated on six years previously. Thoma* points out that follicular cysts that recur after

*Thoma, K. H.: Oral Pathology, ed. 2, St. Louis, 1944, The C. V. Mosby Co.

conservative operation should be examined carefully as all odontogenic cysts are potential adamantoblastomas. Several cases have been reported demonstrating the transition and metamorphosis from follicular or dentigerous cysts to adamantoblastomas.



Fig. 71.—One year after operation. The jaw was strong, functioning fully. A lower partial denture replaced the extracted teeth.



Fig. 72.—Occlusal view one year after operation.

A radical operation was performed, leaving only a thin strip of lower border of the mandible with its periosteal attachment. The excised borders were in healthy tissue. As described by Calvin,* this operation maintains the form and continuity of the mandible and serves as a focus of bone regeneration, as seen in Figs. 69, 70, and 71.

Two years after operation there was no evidence of recurrence. The rebuilt mandible is shown in Fig. 73.



Fig. 73.—X-ray appearance of jaw two years after operation. Compare bone regeneration with Fig. 68.

Case Report of Reconstruction of Lip, Chin, and Mandibular Symphysis

A 24-year-old corporal was admitted to the 6th General Hospital on Aug. 11, 1943.

Chief Complaint.—Loss of bony framework and soft parts of the chin.

History.—Two and one-half weeks before admission this soldier became involved in a controversy in a native café a few miles from Rabat, French Morocco. He and his two soldier companions exchanged gunfire with a native civilian who was reported to have fired twice at the patient at close range—shooting him once in the face and once in the left shoulder region. The wounded soldier was taken by the military police to a near-by general hospital, where his condition was found to be precarious. Superficial débridement of both wounds and general supportive measures were instituted.

He was transferred to the 6th General Hospital on Aug. 11, 1943, with a diagnosis of (1) wound, penetrating and lacerated, severe, of chin; (2) frac-

*Calvin, A. R.: Adamantinoma With Cyst of Lower Jaw, Minnesota M. J. 20: 624, 1937.

ture, compound, comminuted, complete, of symphysis of mandible; (3) wounds, penetrating, multiple, severe, of left shoulder; (4) foreign bodies, multiple, metallic, retained in floor of mouth and left shoulder.

Physical Examination.—On admission, the patient showed a gross defect of the soft parts of the chin and complete loss of the lower lip, with exposure of the floor of the mouth. The denuded, protruding ends of the bodies of the right and left mandible were visible; the tongue, floor of the mouth, cheeks, and chin

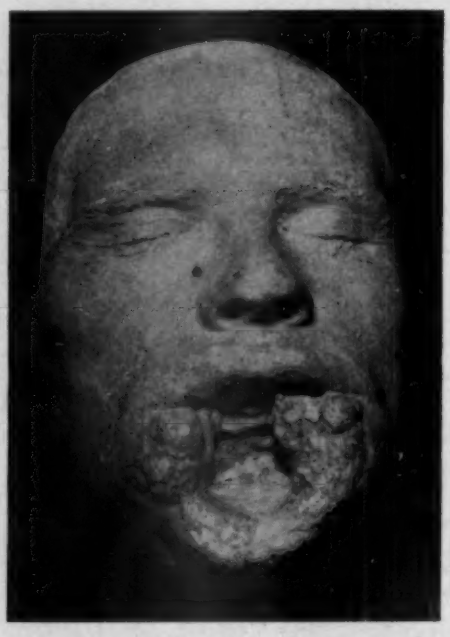


Fig. 74.—Face mask showing loss of lower lip, chin, and anterior section of mandible.

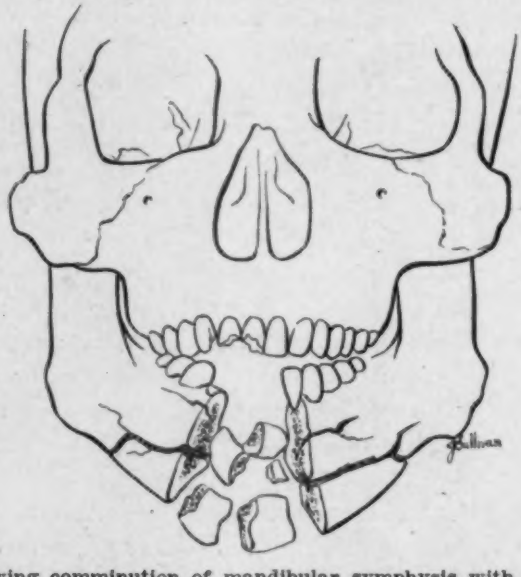


Fig. 75.—Diagram showing comminution of mandibular symphysis with fragments driven into the floor of the mouth. There was medial collapse of the right and left bodies.

areas showed massive edema and some suppuration. There was no fixation of the mandible, and multiple broken teeth and roots were exposed. (Fig. 74.)

The left shoulder, anteriorly, revealed a 5 cm. granulating wound, extending 3 cm. in depth, just inferior to the acromioclavicular joint, surrounded by multiple healed and healing penetrating wounds 3 to 4 mm. in diameter. The motions of the shoulder joint were limited only by pain in the soft part wounds. The patient's general condition was good.



Fig. 76.—X-ray of left shoulder peppered with lead shot. There was no evidence of fracture of the bones.

X-rays revealed multiple comminutions of the mandibular symphysis with at least four major fragments entirely detached and displaced backward and downward in the floor of the mouth beneath the tongue (Fig. 75). There were also multiple pieces of fractured teeth plus metallic foreign bodies driven into the soft tissues of the mouth and jaws. X-rays showed that there were no fractures of the bones of the left shoulder girdle (Fig. 76).

Plan of Treatment.—It was decided to débride and immobilize the jaw in good occlusion as early as possible and to rebuild the lost soft tissue parts by means of a rope graft from the scapula region in conjunction with local flaps

to reform the lower lip. A bone graft to the symphysis region was planned, if necessary, as the final stage after the rope graft reconstruction had healed.

Progress.—Three days after admission, a thorough débridement of the floor of the mouth was done under local anesthesia. The metallic foreign bodies, fractured teeth, and numerous small bone fragments that were completely free of periosteal attachment were removed. Two major bone fragments about 1.5 cm. in diameter were dislodged from the floor of the mouth and brought forward in apposition to the lingual sides of the horizontal rami. These large fragments could not be moved anteriorly as far forward as the labial rim of the missing lower border because of the risk of completely detaching the fragments from their soft tissue bed and also because of the residual brawny edema of the floor of the mouth.

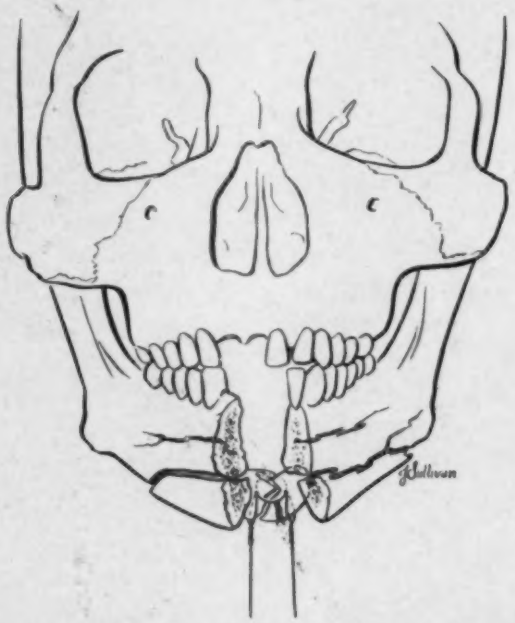


Fig. 77.—Several of the larger fragments were wired together and traction was applied to draw them forward from the floor of the mouth to contact the right and left horizontal bodies.

The jaws were immobilized in good posterior occlusion by means of Kazanjian buttons and intermaxillary elastic traction. The recovered bone fragments were joined by transosseous stainless steel wires. Additional wires were inserted into the drill holes and connected by elastic traction to an extraoral spring steel vertical bar extending from a plaster head cap down over the brow and nose. Thus, constant traction was exerted labially on these displaced symphysis fragments. This held them in contact with the main bodies of the mandible, restoring the continuity of the right and left mandibles. (Fig. 77.)

Next, a carefully measured rope graft was elevated from the region of the left scapula, leaving both ends of the graft attached. The raw surface under the rope graft was covered with a thick split-thickness graft from the thigh.

On September 2, under novocain anesthesia, an additional skin graft was applied to the undersurface of the lower end of the rope to provide the intraoral lining when the graft was swung to the chin.

On September 14, the rope and all free grafts were in good condition, but sepsis about the lower border of the left mandible necessitated incision and drainage under pentothal sodium and postponed temporarily further stages in reconstruction.

The plaster head cap was well tolerated, and x-rays showed the bone fragments were maintaining contact (Figs. 78 and 79).

All brawny edema about the floor of the mouth had subsided under a regime of hot packs, irrigations, and chemotherapy. The following abstracts of progress notes describe future procedures and results:

Oct. 14, 1943: Under pentothal sodium anesthesia, the lower end of the rope graft, completely lined with the well-healed free split graft, was detached from the shoulder region and swung into position on the chin without disturbing traction on the mandibular fragments to the head cap extension (Fig. 80).

Oct. 20, 1943: Rope graft was viable throughout, but the lining of the free split graft became necrotic and was cast off in toto.

Oct. 29, 1943: Head cap and traction on mandibular fragments were removed after ten weeks, with clinical evidence of union.

Nov. 5, 1943: X-rays confirmed the presence of bony union (Fig. 81), and all traction wires were removed. The patient now had a firm lower border connecting the anterior ends of both mandibular rami. A temporary metallic retaining splint from premolar to premolar was fitted for support and the intermaxillary fixation was removed.

Nov. 8, 1943: Under novocain anesthesia the lower lip was reformed by swinging lateral flaps from the cheeks to unite in the midline superior to the attachment of the rope graft. Mucous membrane flaps were at the same time mobilized in a similar position and brought forward and used to reform the lower vermilion border (Fig. 82).

Nov. 22, 1943: Under novocain, the rope to the chin was severed and the proximal part replaced in its bed over the back of the shoulder. The previous plastic reconstruction of lower lip had completely healed with the exception of a small salivary fistula in the midline. The patient was now eating a soft solid diet. The wound of the left shoulder had granulated in and epithelialized, and the patient was rapidly regaining normal motions of his left arm. (Fig. 83.)

Dec. 10, 1943: Under novocain anesthesia, a final, cosmetic procedure on the edges of the newly grafted chin, with removal of superfluous fat, was done (Fig. 84). The salivary fistula had closed spontaneously. The patient was being fitted for a permanent acrylic and silver splint which would not only be removable for cleansing, but also give permanent support for the grafted soft tissues.

Jan. 1, 1944: The patient has shown an excellent recovery; he eats a house diet; all grafted areas are well healed. He has a full range of motion of his left shoulder in spite of the presence of the multiple foreign bodies seen in Fig. 76. He is ready for discharge to duty except that the permanent acrylic splint is not yet finished.

Jan. 22, 1944: The patient was discharged to full combat duty after a little over five months in the 6th General Hospital. With the acrylic prosthesis carry-

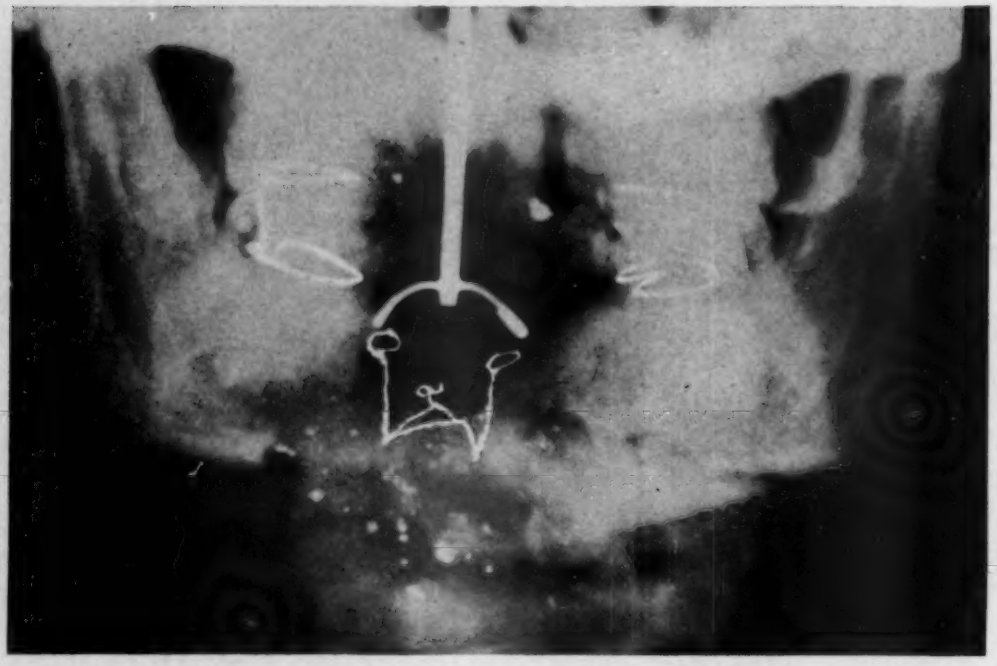


Fig. 78.—Anteroposterior view. Stainless steel wires from symphysis fragments to extraoral spring bar extended from a plaster head cap.

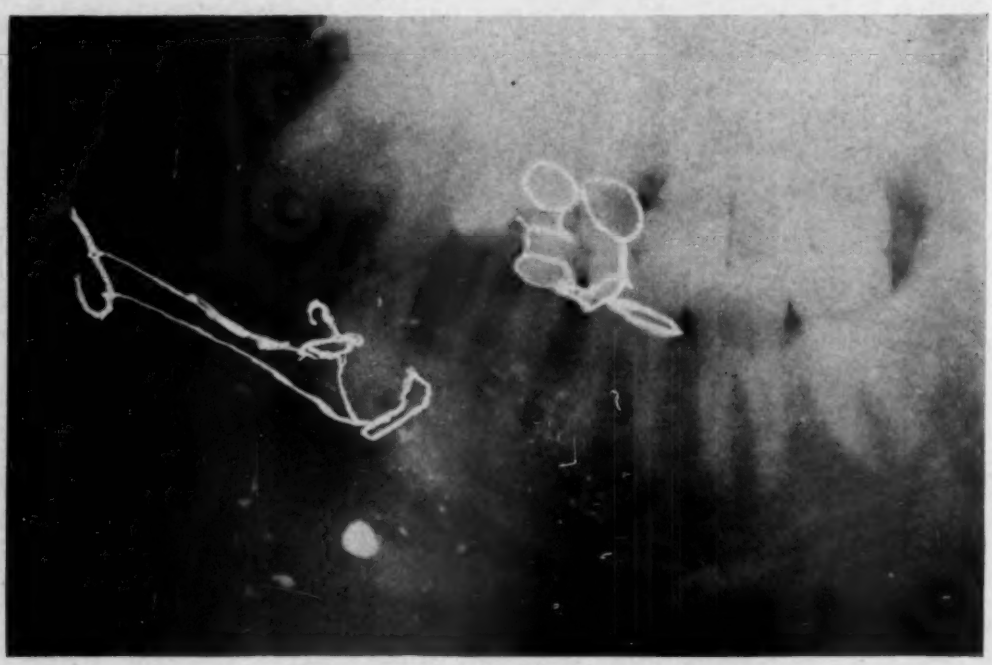


Fig. 79.—Lateral view (wires retouched for clarity).



Fig. 80.—Sketch showing rope graft from shoulder region into position on chin.



Fig. 81.—Occlusal view showing continuity of lower border three months after injury.



Fig. 82.—Sketch showing formation of lower lip by lateral flaps. The vermilion border was formed by intraoral mucous membrane flaps joined in the midline.



Fig. 83.—The donor site for the rope graft to chin. The patient had normal function of the left arm.



Fig. 84.—Photographs showing restored lower lip and chin four months after admission.

ing R 9-19 and L 9-10, he has good occlusion, can eat a field diet, and has sufficient apposition of the lips to smoke and suck on a straw. He has a presentable contour to the chin, although the grafted soft tissues are a lighter shade than the bearded areas of his cheeks. (Figs. 85 and 86.)



Fig. 85.—Lateral jaw x-ray showing bony union in anterior mandible at time of discharge, five months after being shot.

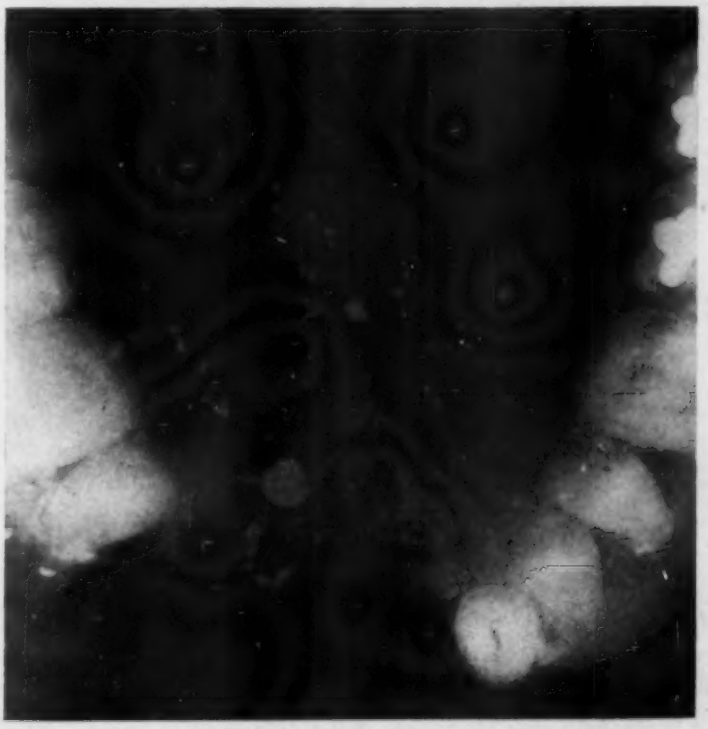


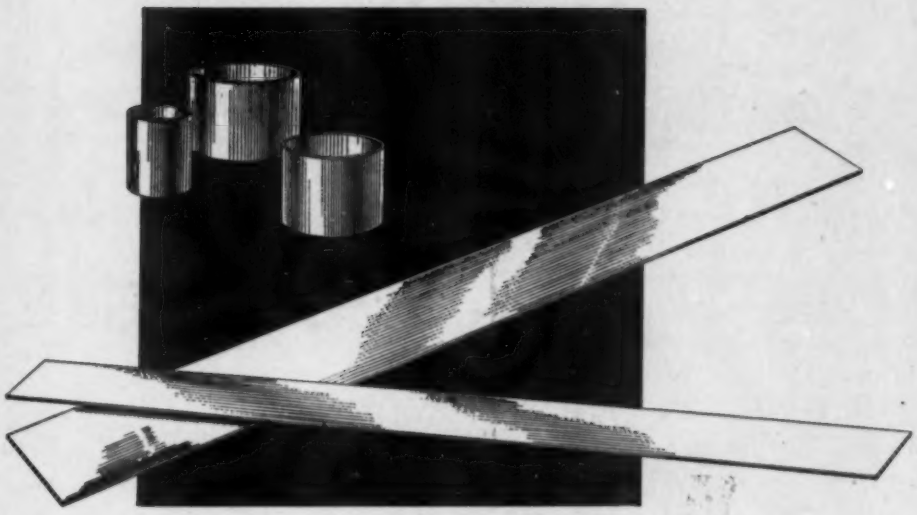
Fig. 86.—Occlusal view showing multiple metallic foreign bodies in the symphysis region. Union was complete and the lower border re-established.

Follow-up Notes.—Nov. 10, 1944. Sixteen months after injury and ten months after discharge, the patient visited the hospital in Rome, Italy. He had seen nine months of full combat duty as a turret gunner in a tank in the Cassino and Rome campaigns. He complained only that his splint had become a little loose and he showed a thin contracted fibrous band inside his lower lip. The color of the chin was sunburned and blended in with the rest of his face. The fibrous band was released under novocain, and he returned to full duty. (Fig. 87.)



Fig. 87.—Face mask of patient sixteen months after injury. He had served nine months of active combat duty since discharge from the hospital.

Summary.—This 24-year-old soldier suffered a major gunshot wound with complete loss of lower lip and chin and fragmentation of the mandibular symphysis. Multiple penetrating buckshot wounds in the soft parts of the shoulder proved to be not incapacitating. Entering the 6th General Hospital within three weeks of injury, a thorough débridement and firm fixation of the jaws was immediately done. These two measures allowed the suppuration and edema to subside so satisfactorily that he obtained solid union of the lower border, reformed by bone fragments, after ten weeks of extraoral traction by transosseous wires to an extension bar from a plaster head cap. In the meantime, a rope graft from the shoulder had been prepared and swung into position, and approximately three months after admission this was permanently in place. Local flaps were used to reform lower lip and vermilion border. It was found that a rigid metallic splint from premolar to premolar would make further bone grafts unnecessary. He was healed and ready for full combat duty, except for preparation of his permanent acrylic prosthesis, about four and one-half months after entry and about five months after receiving this severe and disfiguring injury.



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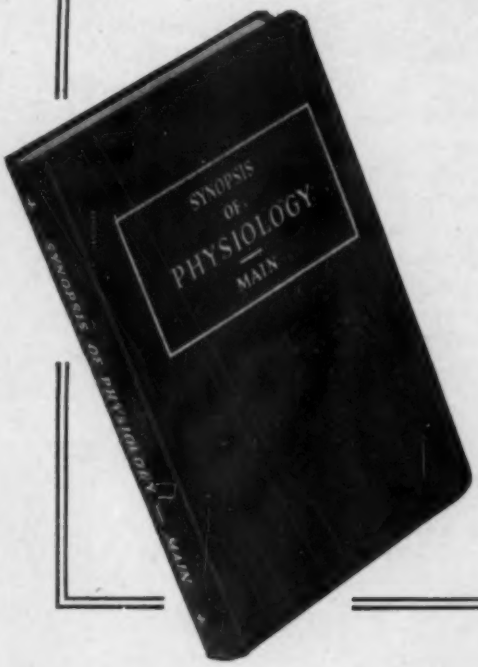
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Cleft Palate Number

THE RESPONSIBILITY OF PUBLIC HEALTH AGENCIES TO CLEFT PALATE PATIENTS

PAUL DODDS, M.D.,* HARRISBURG, PA.

THE figures which Dr. Grace† compiled by actual count of birth certificates allow a more accurate appraisal of the case load than can be found elsewhere. About 275 babies with some degree of cleft palate and lip are born in Pennsylvania each year. This is a sizable load when we consider the complicated procedure and the prolonged care required for rehabilitation.

In the past, the record of salvage has not been good and the outlook of the group to be able to live comparatively normal lives has been discouraging. The results obtained in the rehabilitation of some individuals in the group have been outstanding, but whether a baby born with cleft palate will receive proper care and optimum results seems to depend largely upon chance. There has been no planning for the group as a whole, and the more fortunate ones, the ones who receive all that science has to offer, have by chance or by force of circumstances fallen into the right hands at the right time. These examples of what can be accomplished for the cleft palate patient should give us no peace of mind until the necessary services are made available to all of them.

From a purely public health standpoint, what, up to the present time, has prevented the cleft palate patient from obtaining optimum rehabilitation? In my opinion, the greatest obstacle is the lack of a long-range plan. Formulation of such a plan, I believe, is largely a public health function. This does not mean that public health agencies should concern themselves with delicate, technical decisions, such as the timing of medical, surgical, and dental procedures. Public health agencies should be called upon to set up a total plan whereby our guns may all be brought to bear on the target and fired in a coordinated manner. Only in this way will all cleft palate patients receive care commensurate with our present knowledge.

Undoubtedly, another function in the cleft palate program which ought to be assigned to public health agencies is that of case finding. In fact, at the present time the Pennsylvania Department of Health, through its Bureau of Vital Statistics, its Bureau of Public Health Nursing, its School Division, and its Crippled Children's Division, can report more cases than can be cared for adequately.

Read before the Academy of Cleft Palate Prosthesis, April 10, 1946.

*Director of the Bureau of Maternal and Child Health, State Department of Health, Harrisburg, Pa.

†Linwood G. Grace, D.D.S., of the Pennsylvania Department of Health.

It is unnecessary to point out how useful public health agencies could be in a total program by assisting in the promotion of professional education and in providing nursing and medical social services in the field.

I want now to invite your attention to the financial aspect of a total program for cleft palate patients. Often, but by no means always, the greatest obstacle in the way of adequate care is lack of family funds. This does not mean, necessarily, that such a family is indigent in the strict sense of that term. A family which is perfectly able to take care of itself in other respects might be overwhelmed by the expense of providing adequate care for a cleft palate baby. In fact, when we remember the prolonged and expensive care required, the services of several specialists, the number of operations, and the number of times the child must be hospitalized, *most* families may be considered indigent in respect to the care of the cleft palate patient.

Adequate care should be available to all cleft palate patients and I believe that limitations of the family budget should not be allowed to prevent this. When a family is unable from its own resources to provide adequate care, it is my personal conviction that tax-supported public health agencies and voluntary nonofficial agencies should be called upon to provide the care.

THE RESPONSIBILITY OF THE SURGEON IN TREATING PALATAL AND RELATED DEFECTS

JOHN W. KEMPER, D.D.S., M.D.,* ANN ARBOR, MICH.

PALATAL defects are either acquired or congenital. The incidence of the acquired form, caused by disease or trauma, is very small compared with the congenital form of cleft palate which occurs about once in every 1,000 to 1,200 births. Since the principles involved in the management of the acquired defects are the same as those in the treatment of secondary deformities which sometimes follow primary operations for congenital clefts of the palate, this short paper can be confined in the main to a general discussion concerning the latter.

Rehabilitation of the cleft palate patient may be likened to a great drama in which there are a number of actors, each playing an important role under the direction of one individual, the surgeon, who is responsible for its management and production. The most distinguished and important character, the central figure in the play, is the patient, whose successful performance is dependent on the skillful guidance of the director (the surgeon) and the combined efforts of all the other members of the cast.

Upon the surgeon rest many responsibilities. These begin when he first sees the patient and continue through to the time when the most satisfactory and complete treatment has been carried out, that is, when the patient is able to take his rightful place in society and satisfactorily carry on in our present-day highly competitive way of life. That time may come early in the patient's life, immediately after surgery, but in many cases it extends through childhood and into adult life.

To assume these responsibilities the surgeon must have a full understanding of the other nonsurgical problems essential to his program of complete treatment and at all times give fullest cooperation to those other specialists concerned in these special fields. These special problems must be considered inseparable from his surgical responsibilities. Let us then consider his responsibilities in the order of their occurrence in the life of the cleft palate patient.

The most immediate and sometimes urgent problem is feeding and nursing care during the first few hours or days of the infant's life, especially in those cases of complete cleft of the palate complicated by a cleft of the lip. For the first few days some time may be required in feeding by means of a medicine dropper or small bulb syringe, but, within a few days, the formula can be given from a bottle and nipple with perforations large enough to permit

Read before the Academy of Cleft Palate Prosthesis, April 10, 1946.

*Professor of Oral Surgery, School of Dentistry, and University Hospital, University of Michigan.

the fluid through gravity to drip into the mouth without the act of vigorous sucking. In no case has it been our experience to have to resort to the use of a special gadget, nipple, or prosthetic appliance, or any other complicated procedure to feed the infant.

The next responsibility of the surgeon should be a heart-to-heart talk with the distressed parents and relatives in an attempt to explain: how such a deformity occurs; that it is a frequent occurrence; that the anatomical defect is caused by a miscarriage of Nature's plans and is not a mark of disgrace or infamy; and that the baby, except for this defect, is a normal individual and, if treated as such through their cooperation, in all probability will develop to be a completely normal child. In order to give the parents a more vivid and hopeful illustration of the possibilities of complete cooperative treatment, arrangements can be made for them to see other children who have been successfully treated for a similar or more complicated lip and palate deformity. To arouse hope early in the parents, relatives, and friends of the patient, and to emphasize the importance of their continued cooperation in the plan of treatment outlined to them, is, in my opinion, often a most significant part of the successful management of the whole problem. To assume this responsibility requires much patience and consumes considerable time on the part of the surgeon, who is very often engrossed in a busy surgical practice; yet it is time well spent and often sets the stage for a smooth-running and coordinated program of complete care of the child.

The surgical responsibility, of course, rests wholly with the surgeon, who, after a thorough examination and accurate diagnosis, must decide when and how to operate the case. In making these decisions, many things must be considered, such as the health of the patient, the type and degree and nature of the deformity, the quality as well as quantity of tissue, etc., always with the sole purpose of obtaining a normal anatomical and functional repair. The ultimate surgical result will depend not only on the surgeon's technical skill, but also on his surgical judgment, which he has developed over a long period of specialized surgical training and experience in the management of these cases.

The activity of the growth processes during childhood is an all-important biologic factor which must always be taken into consideration in any surgical procedure attempted at that time. The inherent power of repair of tissues and their response to function are in early life at their highest levels and are essential factors in the creation of normal anatomical design. If any operative procedures are performed which tend to inhibit these physiologic processes, healing will occur slowly or not at all, and there will be little if any attempt toward the restoration of normal structural integrity.

There are numerous sound surgical techniques more or less standardized which are generally accepted and used by most surgeons in palate surgery, but no one can be adopted uniformly in every case. In general, many cleft lip and cleft palate deformities appear similar, yet in most instances there are many anatomical variations which the surgeon must recognize, and through

his experience and judgment he must decide what procedure or variation or modification of various procedures should be used to give the best results in each specific case.

A fairly large percentage of cleft palate patients frequently have an associated cleft lip deformity. It is important that the lip be repaired early (within the first month to six weeks), if the patient's health permits. It is our policy to wait until the birth weight has been regained and the infant shows a daily increase in weight, which is usually about 1 month of age. During these first few weeks of the patient's life, the general condition can be observed and evaluated by the pediatrician, and preparations can be made leading up to the operation. The purpose of early cheilorrhaphy is not for cosmetic effects alone, but in the main it is to obtain normal lip pressure against the protruding or rotated premaxilla in order to encourage, by normal growth process and tissue response to function, its normal alignment in approximation with the maxilla and the closure of the cleft of the alveolar ridge. The growth of the maxilla during the following few months effects a closure of the cleft of the jaw and in most cases gives a normally developed maxilla except for a slight depression or notch of the alveolar ridge where the cleft previously existed.

During the interim between the lip operation and the time to close the palatal defect (which is usually about 2 years of age), cleft palate infants are usually predisposed to upper respiratory and middle-ear infections and should be examined at regular intervals of three to six months, or that responsibility should be turned over to a pediatrician. We consider the optimum time for the reconstruction of the palate is shortly before the child begins to talk freely or has had the opportunity to form faulty habits of speech, which in most instances is between 18 months and 2 years of age.

Fortunately, in nearly every case of this deformity, sufficient tissues are present to permit their rearrangement and fabrication into a finished functioning unit. The nature of the operative program will of course depend upon the degree and type of defect, and the individual preference of the surgeon. Since it is his responsibility to create as near normal an anatomical and functional palate as possible, it is his duty to carry out the surgical procedures which in his experience have given the most satisfactory results. His ultimate aim should be preservation of a normal form to the palatal and dental arches, a flexible musculature, and a palate long enough to permit velopharyngeal closure.

Before beginning surgery it may be well for the surgeon to look ahead and consider his next immediate and very essential postsurgical responsibility and, if possible, to see the patient in consultation with the speech correctionist. A consultative examination by the surgeon and speech teacher prior to the operation permits each to have a better understanding of the other's perspective of the case and effects a more closely coordinated plan of treatment. Normal function may follow surgery, but until the child reaches the age when he talks freely, this cannot be definitely determined. Since normal speech is, unfortunately, seldom a postsurgical reality, a speech training program should

be planned early in every case. If preoperative speech consultation is not possible, it is the surgeon's responsibility to arrange one directly following surgery so that a program of active and passive exercises can be started and carried through until the age when the child can comprehend. Then specific speech training can be instituted. Regular surgeon-speech-teacher consultations should be maintained as long as speech training is necessary.

With the completion of the first dentition arises another responsibility of the surgeon, the dental problem. Dental care, of course, is the responsibility of the dentist. It is, however, the obligation of the surgeon to see that dental care is carried out at the right time and in the right manner. This can be accomplished more effectively when the surgeon has a complete understanding of the existing operative, orthodontic, and prosthetic needs and when he maintains the closest cooperation with the dental specialists.

Secondary operations are not infrequently necessary for residual deformities following an unsuccessful repair of a cleft of the palate. Secondary deformities are in many respects similar to acquired defects and their surgical repair should be considered in the light of an old healed injury.

Small perforations of the hard palate may in some cases be repaired by interpolated flaps of adjacent tissue or the transposition of an extraoral graft. In defects with considerable tissue loss, especially when they involve the soft palate, the advantages of prosthetic assistance should be given just consideration. The surgeon should always keep uppermost in his mind the surgical limitations in many of these cases and should not permit his good judgment to be overbalanced by his enthusiasm and desire to repair by surgery alone. When a prosthesis is indicated for the correction of a defect, either congenital or acquired, he should have a complete understanding of the existing prosthetic problem. By the same token, the prosthodontist should understand not only the involved technical problem but also the surgical, anatomical, and physiological aspects of the case.

A complete study and timely correction of associated secondary facial deformities is another responsibility of the surgeon. Facial disfigurements are occasionally seen following surgical correction of cleft lip and cleft palate deformities, especially the bilateral types. These unfortunate individuals are often as conscious of their abnormal appearance as they are of a major speech defect, and it is of utmost importance that, if possible, normal facial contour be restored in order to avoid the possible added handicap of a psychoneurosis.

The complete care of patients with palatal and related defects carries with it many responsibilities. The greatest responsibility rests with the surgeon who must formulate and organize a plan of treatment and direct its execution. If, under his direction, the highest degree of coordinated and cooperative performance is attained by all those who play a part in this program, the future social and economic welfare of most of these patients will be secured.

PRESENT STATUS OF SURGICAL TREATMENT OF CLEFT HARD PALATE

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THERE are still widely divergent views among oral surgeons regarding the optimum age of the patient for repair of clefts involving the hard palate, and also differences of opinion as to the operative methods preferred in making the repair.

During the past two or three decades there has been a definite trend toward the repair of cleft palate deformities at earlier ages. The consensus at the present time is that—provided the child is in good general condition—incomplete clefts of the hard palate should be repaired some time between the fifteenth and twentieth months of age. In complete clefts of palate, whether unilateral or bilateral, the premaxilla is brought into the best possible position at the time of the primary lip repair, which is usually done some time between the second week and the fourth month of age. Repair of the palatal cleft is usually done between the eighteenth and twenty-fourth month, the definite date depending upon the child's general condition and freedom from upper and lower respiratory infection. Cases in which the palate repair has been completed at these early ages show a higher percentage with good speaking voices than is found in those in which the repair has been longer delayed.

Repairing clefts of the hard palate by the two-stage method greatly increases the percentage of complete closures obtained by the primary operation, and also minimizes the mortality rate. The advantages of the method are that time is allowed for the establishment of adequate collateral circulation in the flaps, and that a child stands two short operations better than one long one. The interval between the stages varies from seven to twelve days, depending both upon the appearance of the flaps and the general condition of the child.

The use of mucoperiosteal flaps in closing clefts of the hard palate is probably the method most frequently employed. However, the use of osteoplastic flaps is definitely increasing, and when the detailed technique of the procedure has been mastered the functional results are most gratifying. This is due to the little disturbance of the muscles of the palate, and to the usually good length of the palate obtained.

Persisting openings in the hard palate in which mucoperiosteal flaps were used in the primary operation, we believe are best closed by utilizing osteoplastic flaps.

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There are very few clefts involving the hard palate which cannot be closed, provided the child has the good fortune to be under the care of a skilled oral surgeon from the beginning of surgical treatment. Cases in which there have been partial or complete failure in union become increasingly difficult with each unsuccessful attempt at repair, because there is necessarily some loss of tissue with each failure, and a poorer blood supply because of the resulting increase in fibrosis. In some such cases it may be wiser to use a plate of sufficient extent to cover the opening when the patient has reached the proper age instead of attempting further surgery.

SUMMARY

The present high percentage of successful closure of clefts involving the hard palate is the result, first, of improvements in operative technique which minimize trauma, and, second, the almost universal adoption of two-stage operations.

When repair of clefts involving the hard palate were repaired by one-stage operations, our better clinics secured complete primary union in 60 to 65 per cent of cases. By the adoption of the two-stage method, complete primary union is now secured in approximately 90 per cent of cases. In the remaining 10 per cent, most of the persisting openings are in wide bilateral clefts and are located just posterior to the premaxilla. The openings are usually closed six to twelve months later by use of mucoperiosteal single pedicled flaps from the extreme anterolateral portions of the palate. If the tissues do not have adequate circulation to make the use of flaps feasible, a partial plate may be used.

135 South 18th Street

SURGICAL TREATMENT OF VICIOUS CICATRICES INVOLVING THE CHEEK, LIP, AND ALVEOLAR PROCESS IN CLEFT LIP AND PALATE CASES

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NOT infrequently, following operations for cleft lip, adhesions occur on the mucous membrane aspect, binding the soft tissues of the lip or cheek to the underlying bone and eliminating or greatly lessening the natural buccal and labial vestibule. These scar bands and adhesions interfere with free movement of the lip and cheek, and also prevent the insertion of proper artificial appliances to restore contour and replace missing teeth.

Simple incision of these bands is generally unsatisfactory, because healing quickly follows, with formation of new scar tissue and aggravation of the deformity.

In a few cases, where the scar is of limited extent, it can be cut through with overcorrection, that is, making the sulcus larger than normal, followed by immediate insertion of a suitable appliance attached to the teeth, with a flange extending well into the newly created space. This appliance must be worn continuously until the raw surfaces of the gap are completely healed, after which the permanent restoration can be placed. As a general rule, however, it is much more satisfactory if, after cutting through the adhesions, the raw surfaces thus created can be covered immediately with epithelium, or closed over in some manner.

In the case of a comparatively narrow scar band, extending across the vestibule from the mucous surface of the lip or cheek to the alveolar ridge, with good mucous membrane on each side, the principle of the Z-plastic can sometimes be used. An incision is made down the length of the band, not across it. From one end of this median incision another incision is made at an angle of about 60 degrees, and about the same length. From the other end another incision is made in the opposite direction, at the same angle and of equal length. This gives a Z-shape to the incisions. The mucous membrane is then elevated from each angle, giving two triangular flaps. The points of the flaps are now transposed and the edges sutured, thus closing all raw areas, altering the direction of the scar line and eliminating the band.

For broad deep scar bands, the Z-plasty will not work. After excision of the scar and overcorrection of the pocket, there remains a large raw surface due to a defect in the mucosa. The deficient mucosa in such cases is best re-

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placed by a skin graft. Skin grafts will heal very well in the mouth and while the skin never turns into mucous membrane, it makes a very efficient substitute. Before the operation, an appliance must be ready to retain the skin graft in position during healing. The most suitable material for supporting the skin graft in the mouth during healing is dental impression compound. After the scar has been cut away and the overcorrected pocket made, softened compound is inserted into the pocket, allowed to harden, and then removed. This gives an accurate impression of the pocket. A rather thin skin graft, with raw surface out, is then draped over the compound mold, and the mold, with graft covering it, is reinserted in the mouth, being held in place by an appliance most suited to the individual case. Sometimes a bridgelike appliance attached to the teeth is provided with a removable wire extension which is embedded in the softened compound, the removable piece being bolted to the fixed part of the appliance after the graft has been inserted.

Frequently, when an artificial denture is to be provided, this can be used as a support for the skin graft by building compound onto its vestibular flange to fill the new sulcus. The plate can then be inserted after the compound extension has been covered with a skin graft. Skin grafts tend to undergo considerable shrinkage in these pockets unless held for a time under tension, and the object of the operation would be defeated if the desired size of the pocket were not maintained by keeping it filled with compound for several weeks after the operation. The first piece of compound should not be disturbed for at least ten days, after which it may be removed for cleansing the parts and inspection of the graft, which by that time should have become adherent to the underlying raw surface. The original compound or a fresh piece should then be reinserted for two or three weeks more. After this, a permanent extension of acrylic or vulcanite can be worn.

The procedures outlined above are applicable not only to cleft lip and palate cases but to adhesions and scar formations diminishing the buccal and labial vestibule as a result of injury, operation, or disease.

1930 Chestnut Street

THE RESPONSIBILITY OF THE ORTHODONTIST IN THE CLEFT PALATE PROBLEM

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IN ORDER to discuss this subject properly it is first of all essential that we agree on the category in which the cleft palate patient belongs. Before either the orthodontist or any other member of the allied professions can assume a responsibility, it is obviously necessary to arrive at some conclusion as to their particular individual liability. At the present time the problem of orientation seems to this essayist to be the confusing issue and when once clarified should immediately simplify and expedite the solution of our individual problems.

In 1840 when the first dental college was founded, separating the training of dental students from medical students, some factors of greater import took place than they at that time ever dreamed could happen. I am referring specifically to the place dentistry occupies in the field of public health and its position in relation to those problems. For those results culminating from the independent stand of dentistry were long and far-reaching. For too many years the importance and need of good dentistry were forgotten factors in the public health program. One has only to take the statistics compiled from this last war to realize how devastating that separation became and how completely overlooked was the part dentistry must play. When one considers the dental situation generally and realizes that in the Army alone approximately 3,000,000 dentures were made for our young men, the cream of American manhood, the ones who were our school children only yesterday, one sees the significant fact that dentistry had been neglected by those responsible for our public health structure.

At the present time clinics are established in the United States for practically every ailment to which the human being falls heir, but the figures resultant from this last war show a poor solution to our dental problems, completely out of proportion to the magnitude of the needs. So in like manner in the field of orthodontics this separation in thinking has produced still more startling conditions, precluding any satisfactory solution of its problems and hindering its function in fulfilling public health demands.

What is orthodontics? Is it not dental facial orthopedics? If so, should not the crippled children's programs include the service of orthodontics? Or are the facial cripples to be neglected? Until a place can be found to orient these unfortunates properly, nothing can really be assumed as a responsibility

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by any one branch of the healing arts. In a paper[1] published in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY in 1942, I wrote as follows:

> As time goes on we are more and more frequently impressed with the realization that although orthopedics and orthodontics are so closely related, yet in the practical application of treatment of deformities, orthodontics and orthopedics are completely severed. It seems unusually singular that although we have such a great common meeting ground in Wolff's Law, nevertheless one is forced to face the question with alarming frequency, "Why cannot the treatment of facial deformities be carried out as any other branch of orthopedics?"

In attempting a solution to the problem of how and where these needs are to be met I continue:

> To help a facial cripple properly and serve him scientifically requires just as much of a well-rounded dental staff as is required by the orthopedist. When we consider the need of the worst of these cases, the cleft palate patient, we face such a severe deformity and extreme condition that the task is tremendous; and a successful result can be accomplished *only* when given the proper opportunity and assistance. An adequate dental staff should consist, first of all, of a qualified oral surgeon; a dentist who will do the best dentistry possible for these patients; a trained orthodontist; a prosthodontist especially taught for this department; the speech correctionist and finally the psychologist. This group working together is just as important as those before mentioned in the general orthopedic department and is essential to the smooth functioning of successful treatment.
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> This is not a plea for socialized treatment any more than we can call the present orthopedic setup socialized medicine. But it is a plea for a clear look into the future; to see to it that state funds and private funds set aside for the treatment of general deformities are also made available for those involving facial deformities; that eventually our facial cripples will get the same consideration and on the same basis.

It has always been very difficult to understand why we continually hear the statement made that there are no funds set aside for the treatment of these facial deformity cases. Evidently they are not considered true deformities, for we do not require separate funds to treat a right or left arm nor separate funds for a leg or a back. It seems singular that any deformity which requires treatment by an orthodontist must come from a particular fund for that purpose and very rarely is there any available. This, to me, is the greatest illustration resulting from that long ago event in 1840 when we separated the training of the dental student from that of the medical student. It seems we now try to separate the body because of that confusion in our thinking.

In order to illustrate let us consider Figs. 1 and 2.

Fig. 1 shows a boy with anterior poliomyelitis. That disease is in reality a disease of the nervous system, and because of it the innervating influence to his one leg was disturbed. Consequently the leg has become deformed causing him to become a "crippled child." In Fig. 2 we have a boy with an early temporomandibular ankylosis. According to Thoma and others, we know that the growth of the mandible is influenced more by muscle function than is the maxilla. It has been demonstrated that the growth of the maxilla is influenced by atmospheric pressure, whereas in the mandible, muscle function is the greater factor. But in both cases the same fundamental causative factor exists

to produce a definite deformity. If the boy in Fig. 1 is a crippled child, what are we to call the boy in Fig. 2? In the United States today we have funds set aside for crippled children's programs which adequately care for the leg of the one boy, but except in a very few instances completely overlook the jaw of the other boy. So also in studying textbooks relating to general bone deformities we have noticed the space devoted to the cause and treatment of conditions, but, except for wryneck, no reference is made to any of the malformations of the jaws and facial bones. One could almost ask the question, "When is a crippled child not a cripple?" And be answered immediately with, "When he has the deformity above the neck."



Fig. 1.



Fig. 2.

Fig. 1.—Lack of development of left leg resulting from anterior poliomyelitis. (Whitman: Orthopedic Surgery, by permission of Lea & Febiger.)

Fig. 2.—Lack of development of the mandible resulting from temporomandibular ankylosis. (Courtesy Dr. Kurt H. Thoma.)

To return to the definition of orthodontics, Angle in 1900 defined orthodontia as that science which has for its object the prevention and correction of malocclusions of the teeth. Strang[5] later elaborated on that by this statement: "No orthodontist can hope to practice his profession in an intelligent and efficient manner until he has grasped this vision in all the bigness and broadness of its true meaning. . . . This field is not a limited one by any means but reaches out to touch and influence the entire range of facial anatomy as well as that of the neck and the thorax." Salzmann[4] states in his recent book: "Orthodontics comprises the science and art which deal with the developmental and positional anomalies of the teeth and jaws. . . . Orthodontics is based on anthropology, anthropometrics, phylogenetics, biometrics, gnathostatics, endocrinology, pediatrics, dietetics, clinical medicine and dentistry, and most if not all of the basic biologic medical and dental sciences." Orthodontics

also has been called dental facial orthopedics, and until we eventually arrive at some conclusions as to what position dentistry in general and orthodontics in particular will occupy in health thinking, it is difficult to discuss the responsibility that the orthodontist must assume as his in the complete program.

That the orthodontist must assume a great share of responsibility in the treatment of cleft palate cases is assuredly a fact. Textbooks have been mentioning this, but the present general setup for handling of these cases has been so insufficient and poorly coordinated as to discourage most orthodontists who felt the results obtained were not worth the effort. The orthodontist as an orthopedic dentist, by training in biological sciences and by his use of appliance technique, should be in an ideal position to recognize the needs and treatment necessary for these people. In fact, I have often felt that just as in the orthopedic hospital the head of the staff is the general orthopedist, so the day should eventually arrive when the head of the dental staff in that same institution will be the orthodontist. I will admit that the present state of orthodontics is not up to this idealistic attitude, but the future of dentistry as a profession depends upon some of its specialties finally assuming more than the role of technicians.

Since orthodontics is the study of the growth and development of the face and jaws and the functions relating to these structures, it surely seems that the responsibility of the orthodontist is extremely great and challenging. The orthodontist of the future by virtue of his training and interest will have to consider it his duty to assume more responsibility and be willing to strike out and lead in these matters. Many cases would be immeasurably improved in later years if we had only attempted to do long ago what we are trying to do at this meeting today. To see how far surgery will and can go, and to make sure that the orthodontist continues the treatment with the feeling of closest cooperation will enable the two specialties to accomplish so much more than has been done.

In considering the establishment of separate clinics for the treatment of cleft palate patients it seems only logical to include those of other great facial deformities. I feel that such cases are an equal responsibility but one which the orthodontist hesitates to assume before he has been authorized to do so. All deformities of the face or teeth, those either with or without a speech and psychological problem, would loom as equal in importance with the cleft palate.

Fig. 3 shows a case of a boy with cleft palate with resultant facial deformity. He has a speech and psychological problem.

Fig. 4 shows a case of extreme mutilated distoclusion. His deformity is also great. His speech and psychological problem, although not quite as severe as the boy in Fig. 3, is still obvious. How are we going to separate these two boys so far as treatment in a clinic is concerned?

In the Lancaster Rotary Club Cleft Palate Speech Clinic we are constantly faced with such patients. They are for the most part patients with unusual deformities and speech problems who quite confidently apply for service, thereby putting us in a most peculiar position. At one time we are criticizing the

handling of cripples generally and know and feel that a great group have been discriminated against, and yet we turn about and advocate, if we are not careful, the same thing in our facial deformity cases. We cannot separate these people. By what manner of logic do we take a person who has a cleft palate with a speech defect and refuse a patient with a disfiguring distoclusion or mesioclusion deformity which very often presents a speech and psychological problem also?



Fig. 3.



Fig. 4.

Fig. 3.—Deformity resulting from cleft palate.
Fig. 4.—Deformity resulting from distoclusion.

Six years ago a baby boy was referred to me who suffered from a partial absence of the tongue. We have watched the case closely through the years to the present time. His tongue has grown some but not nearly enough to stimulate proper development of the mandible which is also the lower border of his face. His speech naturally is influenced by this and is the reason for his presentation today for your consultation and discussion.

Another very rare case was referred to our clinic, a patient with cleft palate and harelip associated with complete anodontia. The deformity is terrible and will grow worse as the child develops. We hope to present a complete case report of this patient at some future time. Suffice it here to say that there are many more cases of other facial deformities that are constantly applying for treatment. It is difficult to provide treatment for the cleft palate patient with or without a speech and psychological problem and neglect the other patient whose deformity and psychological handicap is just as great. The program must be all-embracing.

We are handicapped in our clinic very often because cases come to us too late for ideal treatment. Too often compromise treatment must be undertaken. Surgery has been done for them, but that represents the sum total of the treatment received. I hope I will not be misunderstood on making the statement that too often the surgery was only mediocre, usually done by one

trained in abdominal surgery, with aptitude or special training in this particular field. Here also is represented a very severe problem, for where poor lip surgery has been performed the orthodontist is given no chance to produce good facial contours. There must be the closest cooperation between the orthodontist and the surgeon for we know that proper muscle pressure is a necessary part of treatment for a final permanent result. I am not now referring to the artists, the trained, scientifically minded surgeons, represented here today. I am referring to the great group of general surgeons who have been doing these operations in their respective communities because they felt there was nothing to do but make the attempt. The fault probably lies even deeper in the fact that there has never been any program outlined and established to orient these patients and place them in their proper category.

Dr. Linwood Grace[2] in a statistical study states that 1 out of 800 live births suffers from a cleft palate. Using the same statistics of 1 in 800, the ratio of poor people in that group is bound to be high, so usually it becomes necessary for the parents to resort to charity, which in turn forms the reason for the attempt by the abdominal surgeon. It is the best surgery available under the circumstances.

The orthodontist, too, is greatly handicapped in his treatment of cleft palate patients because they have not been sent to him at an early age. In the Lancaster Rotary Club Clinic where the cost of extensive treatment would not prohibit the ideal handling of a case, the time element proves to be a problem. There the wisdom, experience, and judgment of the orthodontist must stand him in good stead, for sometimes a compromise treatment proves to be the best. The proper cleft palate treatment requires a thorough over-all view of the problem and in some cases it is obviously impossible to take one, two, or three years for orthodontic treatment when the *total* rehabilitation problem of the patient is demanding immediate attention. The orthodontist must decide when the compromise is justified for the problem at hand. The short treatment can be done with operative dentistry in the form of jacket crowns, thimble crowns, etc., plus unusual prosthetic measures, so that the speech clinician and the psychologist can work with us in helping to solve the total rehabilitation of the patient.

If a program is eventually developed in which these people who have been operated on as infants can be constantly contacted and watched so that proper dentistry will be done on the deciduous teeth and orthodontic treatment given at the proper time, then and then only will the orthodontist be able to function in his ideal capacity. On the other hand, we see many cases in our clinic where orthodontics is of paramount importance and where treatment can be accomplished successfully in a very short time. Models of one of these cases are demonstrated in Figs. 5 to 9.

Figs. 5 and 6 show right and left view of the malocclusion still remaining after much lip and palate surgery was done. There still remains an opening on the left side well into the palate connecting with the nose.

Figs. 7 and 8 show the case after orthodontic treatment.



Fig. 5.



Fig. 6.



Fig. 7.



Fig. 8.

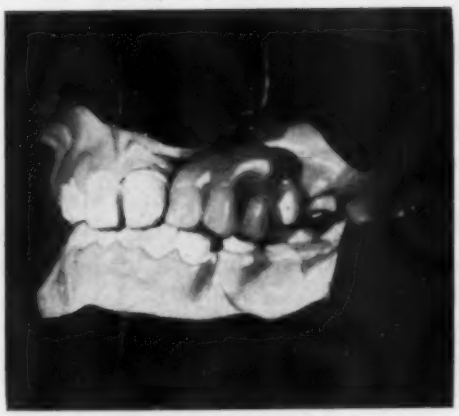


Fig. 9.

Fig. 9 shows the restoration that carries a soft acrylic attachment to make a seal of the opening into the nose. It also carries an acrylic obturator into the pharynx. This restoration has improved the patient's occlusion and appearance. It also acts as a retaining appliance. She is having speech retaining and psychological adjustment help in our Rotary Club Clinic at the present time.

Of the cases we have treated there are some in which the upper anterior teeth still remaining are in lingual version to the lower anteriors similar to that shown in Fig. 5. These can be brought over the bite into proper occlusion and become a great aid in establishing facial harmony and better speech. Here a word of warning is very necessary for we have found that in types 3 and 4 cleft palate patients where one or more of the teeth are in close proximity to the cleft, the problem of retention is so severe as to preclude any possibility of a good result. Therefore we have been removing such teeth and using artificial substitutes because relapse means more than the loss of teeth in the greater danger of holding back the entire program for the patient's total rehabilitation. This conclusion and decision have been reached after various experiences in the past years in which we found ourselves too concerned with a tooth instead of a total personality.

There is another type of case which presents a full complement of teeth in the mandible but due to the cleft palate will never get the same number of teeth in the maxilla (Fig. 3). Here we have best illustrated one of the forces of occlusion, namely, harmony in the size of the arches. So from the orthodontic standpoint we feel that the compromise treatment of the removal of a lower premolar on each side of the arch helps us to produce another deformity to conform to the original deformity which in turn produces an illusion of a better facial balance. In the more adult patient we do not feel that it is always advisable to do extensive orthodontic treatment where the danger of relapse is too evident, because, should the relapse take place, the entire program is greatly retarded and in many instances the results are entirely defeated. We must realize we are dealing with human beings who must be educated and inspired to persevere in achieving the results we now dream of for all the patients.

In conclusion, I might remind you of Dr. Wm. J. Gies' report[3] following his study for the Carnegie Foundation, for this statement, though well known to all of you, bears repeating: "Whether dentistry now is or ever will be a branch of medicine is not the point, but that dentistry is a part of public health of equal importance to the respective branches of medicine is a fact." We have not yet accomplished too much to justify that statement but we should never cease trying to make it known by action. Our potential strength in the public health picture is stupendous if used properly with the complete cooperation of all the allied professions. If dentistry continues to be too highly a specialized and separated group and, within its boundaries, orthodontics a still more specialized unit, a danger lies in the fact that we may lose sight of the complete picture of our public health worth.

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THE RESPONSIBILITY OF THE PROSTHODONTIST IN THE TREATMENT OF THE CLEFT PALATE PATIENT

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THE successful rehabilitation of the cleft palate patient presents a scientific and humanitarian challenge of very great proportions. So complex an enterprise requires the integration of the most mature knowledge, judgment, and experience. In the consideration of our individual responsibilities in this cooperative task, we can best be guided by a very simple statement of fact: the cleft palate patient is, first of all, *a person*. He is not merely a surgical problem. He is not to be viewed as a dental problem. He is not to be described as a speech problem. Nor is he a psychological problem. He represents more than these. He is a person who for a great many reasons finds personal and social adjustment very difficult. Expressed more fully, he is a person who is unhappy, uncomfortable, and unhealthy. He is gravely handicapped in his competition with normal persons in earning a living; he is at a serious disadvantage in winning the respect of his associates, and he is discouraged from forming close and rewarding friendships. To view him merely as a patient with a problem that lies exclusively in our specialty is to lose an important perspective in evaluating the significance and value of our individual efforts.

I am convinced that the prosthodontist is under the severest obligation to appraise the results of everything he does in terms of the searching question: What will this achieve for the person? Such a criterion will demand that the prosthetist be not only willing, but determined to possess a full knowledge of the basic anatomical and physiological facts that relate to the area in which he works. More than this, he should see the necessity of gaining an adequate knowledge of the collateral areas in physiology and anatomy. This will help him to see the complete functional implications of his work. This implies more than a cursory knowledge of the superficial structures of the mouth, nose, and throat; it means, for example, that he must come to feel at home in the more subtle details of the myology and osteology of the upper respiratory and alimentary tract.

But even this is not enough. The thoroughgoing prosthodontist will also acquaint himself with the important facts involved in speech production. Since he presumes to assist in the reconstruction of the parts that subserve the speech function, then it is his responsibility to attain a working insight into its mechanisms. He should not complain that such study is alien to his primary interest and tasks. Inquiry into how the body produces sound for communication is as imperative and logical as is the inquiry into the details of mastication and deglutition. What could be more pertinent than the facts that underlie the principal function he seeks to restitute.

Read before the Academy of Cleft Palate Prosthesis, April 10, 1946.

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Further, the prosthodontist must come to have a good grasp of the descriptive pathology of nonunions of the lip and palate. He must be able to express fluently the facts of this pathology and to understand their relation to his clinical methods and devices. To understand palatal pathology is to be made fully aware of the extent and nature of the functional disorder created by this arrest of development.

There is real danger that the knowledge and the clinical experience in the treatment of the cleft palate patient may become departmentalized. Obviously, the person whom we undertake to help cannot be so fractionated. Certainly the outstanding feature of the rehabilitation of the cleft palate patient is the demand it makes for the cooperation of a number of related and interdependent specialties. In certain cases of cleft palate and cleft lip the prosthodontist expects that antecedent surgery shall be performed with both technical competence and insight. It would be wrong, however, for the prosthodontist to anticipate such surgical ability and result without a full appreciation of the problems characteristic of surgery. Such a recognition of the advantages, limitations, and difficulties of surgery can come to the prosthodontist only if he is willing to make a careful and systematic study of the history and development of the various surgical approaches to cleft palate. On the other hand, the prosthodontist is justified in the expectation that the surgeon will be equally willing to come to a full knowledge of the points of view and techniques of the prosthetist.

The rehabilitation of the cleft palate patient does not consist of choosing this method *or* that method. Rather it is the proper emphasis on the method most appropriate to the particular case.

The intimacy of interest between the orthodontist and the prosthodontist certainly does not need to be underscored here. In the practical situation it is often necessary for the orthodontist to evoke certain essential changes in the deformed dental arch as a preparation for a satisfactory prosthetic speech aid. From a clinical standpoint, however, it is equally true that often a much wiser procedure is to omit *extensive* orthodontic treatment, and to produce a prosthetic correction that fully compensates for the structural variations and allows them to remain. In younger patients the goals of the prosthodontist and the orthodontist are especially companionate.

It is important to emphasize that it is also in the interest of the patient, that the speech correctionist and the psychologist share the responsibility of becoming acquainted with the major technical problems facing the prosthodontist and the methods he employs.

The prosthodontist looks to the speech clinician for highly exact guidance. But such guidance can come only from one who takes the pains to learn what the prosthodontist thinks and how he works. Conversely, the prosthodontist must be aware of the problems of reeducating the patient to speak with a mechanism that is both disordered in structure, and employs a prosthetic device. The prosthodontist should remember as he studies his patient, plans, constructs, and fits his appliance, that he is not merely "filling a hole" in the mouth. Rather he is providing a means by which the cleft palate person

can do what a normal person is expected to do. He must come to view the instrument he creates as a means to an end and this end is an improved personal and social adjustment.

It is unfortunate that the practice of general dentistry does not make practical the acquisition of certain special skills that are essential in satisfactory cleft palate prosthesis. If the general dentist does undertake such work, he must consider it implicit in his decision that he familiarize himself with these special techniques and practice them sufficiently to be able to produce a functional and comfortable speech aid.

It will not be amiss to review in some detail the preparation the prosthodontist should make for the study of the case at hand. Without the complete understanding of his patient that comes about by such preparation, he is likely to be guided by *mechanical* and *technical formulas* rather than the individual *needs* of his patient. His case record should include a description of the original pathology, and the complete history of surgical intervention from the best possible informants. He should take the pains to record exactly the condition he finds, and his impressions of the patient's reactions to them. Into this record should go a summary of the patient's intellectual and emotional status, a phonetic analysis of the patient's speech, an audiogram, a phonograph recording, and extra- and intraoral photographs. A detailed statement of the condition of the mucous membrane and the teeth should be included. There should also be in this record a description of the patient's occlusion and any other information of dental significance. This record he should study.

Thus armed with a full and detailed understanding of his patient's condition and his needs, the prosthodontist at once assumes full stature in the partnership of the joint consultation.

We have arrived at the place where the prosthodontist must prepare himself to be something more than a dental consultant. He may insist that he be granted a place of equivalence in the cooperative scheme of clinical care for the patient. The prosthodontist is also prepared to share the responsibility of deciding with his surgical and speech associates what type of correction should be employed. It may appear presumptuous, but it is nonetheless true, to say that prosthesis is not necessarily a method of second choice. It is one of several methods equally deserving of consideration and study. It is eminently unfair to the patient to deny him the right to *all* avenues of help. Unfortunately, it is still regarded as a dictum that prosthesis should be employed *only* if surgery has failed. Unbiased clinical experience does not support this view. It is a sobering observation that surgery is irreversible. Its injudicious employment more often precludes the most satisfactory use of prosthesis than should be the case if we take our responsibility to the patient seriously.

But there is another urgent responsibility of the prosthetist. This is to build an appliance that is mechanically and functionally satisfactory. Nothing is gained by pointing out the fact that surgery sometimes fails. This does not remove the obligation of prosthesis to prove itself to be a method of choice in the appropriate case. I am confident that surgeons will not continue to

insist that surgery excludes any other approach when more patients are fitted with functionally satisfactory appliances. On the other hand, it is a responsibility of the prosthodontist not only to produce successful speech aids, but to insist that clinical integrity requires that prosthesis be given equal advantage in the joint effort to help the patient. It is not to be denied that prosthesis has special obligations in the cases of postsurgical failure. But this should not obscure the fact that prosthesis may well be the initial and only method of helping cleft palate patients. But neither is prosthesis a panacea. It, too, can and does fail, and such failure is serious for the patient.

There might be considerable profit in detailing here what the criteria of a good functional speech aid are:

1. It must be so shaped and so located that the remnants of the divided soft palate together with the associated muscles can regulate efficiently the pneumatic and acoustic traffic in the nasopharyngeal port.
2. It must be designed so that it has proper retention, bracing, and support to achieve a balanced functional occlusion.
3. Where necessary, it must assist in accomplishing an esthetic improvement.
4. It should improve the oral and nasal hygiene.

We may also be guided by listing the conditions in which prosthesis may well be employed as an equivalent or preferential route of choice:

1. In cases of complete unilateral and bilateral clefts that have been closed anteriorly by surgery but in which the musculature of the nasopharynx and oropharynx is undisturbed.
2. In cases of incomplete cleft of varying degree.
3. In cases of postsurgical accident or failure in which sufficient functional muscular tissue remains to take advantage of the principle upon which a speech aid operates.
4. Cases of postsurgical accident or failure in which there is extensive loss of tissue.
5. Cases of postsurgical accident or failure in which extensive cicatrization renders the muscular tissue immobile.
6. Cases of acquired clefts.

It should be observed that the prosthodontist cannot escape the responsibility of hastening the day when surgery and prosthesis may, by preplanning, be used in the *same case* and complement each other. Such an idea is not new.

The achievement of a goal so beneficial to the patient can be encouraged best by a basic attitude of adventure and exploration in the prosthetic profession. It can be stimulated by the prosthodontist's recognition and admission of his indebtedness to earlier and contemporary workers. The histories of surgery and prosthetic treatment of cleft palate share one very common quality. It is that each operation and each prosthetic device has profited greatly from others that have gone before.

Finally, prosthodontia can expect to increase its contribution to the science and art of rehabilitation only as it evidences persistent interest in developing new methods, finding new materials, and maintaining an experimental and open-minded attitude toward its tasks.

OBTURATOR OF CHOICE FOR CONGENITAL CLEFT PALATE

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ABOUT one out of every thousand children is born with a cleft palate; therefore, every practitioner may find himself in a position where he has to make an obturator. It has to be understood, though, that before proceeding to make an obturator he should consult a surgeon and a logopedist. For psychological reasons, too, the obturator as such may only then be the method of choice if an operation appears impracticable on anatomic or physiologic grounds, or if the operation is not permitted for some reason or other. An obturator is also indicated after unsuccessful operations, and in order to improve the pronunciation after operations, when reoperation has no prospect of success.

This paper deals exclusively with the prosthetic treatment of congenital cleft palates. Its first part is purely anatomic and physiologic. The second part is concerned with the various kinds of obturators, their construction, and their relative value.

ANATOMIC

According to the classical description of Luschka (1864) and Pickerill (1912), the muscles of the soft palate can be divided into three groups on either side: One pair ascending from its posterior edge (levatores and tensores palati); one pair descending (palatopharyngei and palatoglossi); and two each leading from the medial front toward the rear (azygoi uvulae).

1. The two former pairs of muscles form U-shaped loops with their fellows of the opposite side, their horizontal parts leading through the soft palate.

The levatores palati originate from the tip of the pars petrosa of the os temporale and from the inner surface of the cartilaginous part of the tuba eustachii; they are thus coming from the upper rear. These muscles raise the velum palati from both sides and pull it slightly backward, causing the rim of the vellum to get into close contact with the posterior pharyngeal wall.

The tensores veli palatini originate almost at the same place as the levatores, viz., at the fossa scaphoidea, the spina sphenoidalis, and the cartilaginous part of the tuba eustachii. But these muscles press the velum downward, thus causing a tension of the velum, as their tendons wind round the hamulus of the processus pterygoideus.

2. The palatopharyngei form the posterior pillars of the isthmus faucium. The ascending part of the palatopharyngeus, the pars thyropalatina—arising from the posterior rim of the cartilago thyroidea along the lateral walls of the oral pharynx through the arcus palatopharyngeus toward the soft palate, where the fibers cross each other and finally disappear—forms with its opposite number an open U. To this, however, a circular muscle, the pars pharyngopalatina, is attached—originating in the aponeurosis palati, descending in the arcus palatopharyngeus toward the posterior pharyngeal wall, where the fibers diverge and meet with the partner of the opposite side. Thus, the velum can get into direct contact

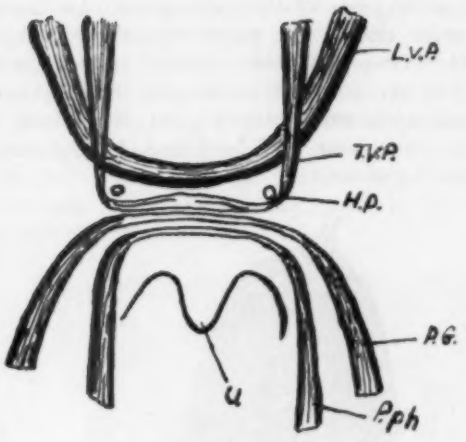


Fig. 1.

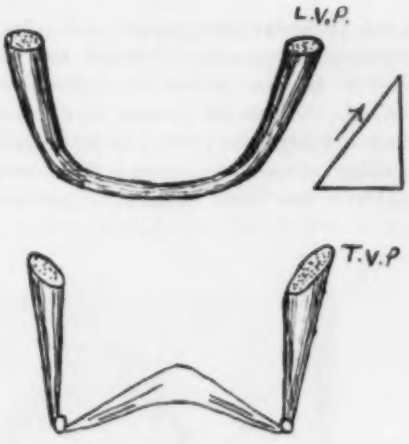


Fig. 2.

Fig. 1.—Diagram of the muscles of the soft palate: *L.V.P.*, levator veli palatini; *T.V.P.*, tensor veli palatini; *H.P.*, hamulus pterygoideus; *P.G.*, palatoglossus; *P.Ph.*, palatopharyngeus; *U.*, uvula.

Fig. 2.—The levatores veli palatini with diagram of their action, and the tensor veli palatini.

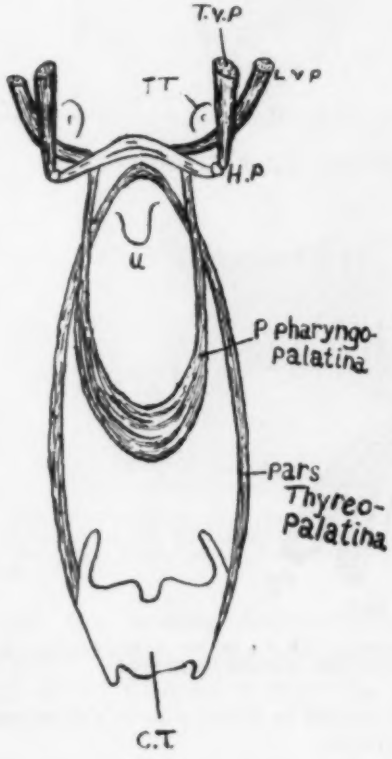


Fig. 3.

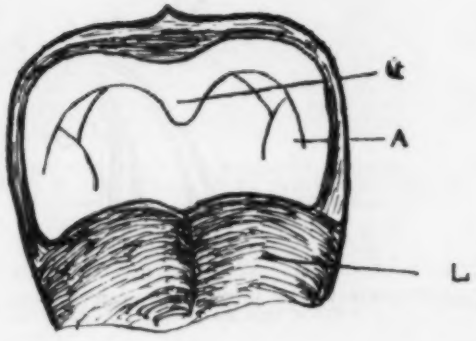


Fig. 4.

Fig. 3.—Diagram of the palatopharyngeus: *T.V.P.*, tensor veli palatini; *L.V.P.*, levator veli palatini; *T.T.*, torus tubarius; *H.P.*, hamulus pterygoideus; *U*, uvula; *C.T.*, cartilago thyroidea.

Fig. 4.—Diagram of the palatoglossus: *U.*, uvula; *A.*, tonsilla palatina; *L.*, lingua.

with the posterior pharyngeal wall. The ascending parts of the palatoglossi pass through the arcus palatoglossus. Through the juncture of their lower parts with the tongue, the inverted U becomes a real circular muscle. Therefore, both these muscles draw the velum downward, or even the tongue upward, whatever the state of tension in the neighboring muscles. If both the levator and the palatopharyngeus are contracted, the components that are pulling upward cancel each other out, while those that tend backward, by combinative effect, draw the velum toward the posterior pharyngeal wall.

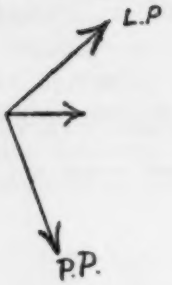


Fig. 5.



Fig. 6.

Fig. 5.—Diagram of the resultants from simultaneous action of the levatores palati and pterygopharyngei.

Fig. 6.—Diagram of the constrictor pharyngis superior (also pterygopharyngeus).

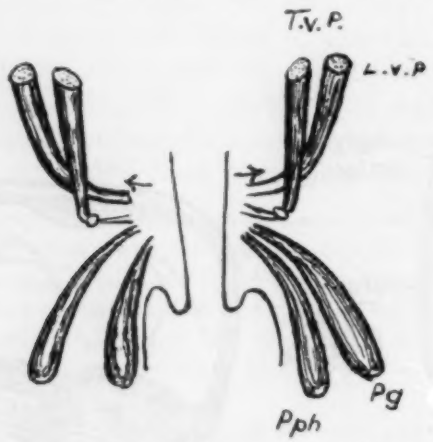


Fig. 7.—Diagram of the muscles in case of cleft palate: *T.V.P.*, tensor veli palatini; *L.V.P.*, levator veli palatini; *Pg*, palatoglossus; *Pph*, palatopharyngeus.

3. The azygoi uvulae are a pair of cylindrical muscles on either side of the medial line of the soft palate. They shorten and condense the velum.

The constrictor pharyngis, too, is U-shaped; like a horseshoe it leads from the processus pterygoideus, the hamulus, and the tendinous continuation of the raphe perimandibularis to the posterior pharyngeal wall. Within and in front of its semicircle lies the sphincter part of the palatopharyngeus.

In case of a cleft palate, therefore, the cleavage ought to broaden considerably with every contraction of these U-shaped muscles. Observations on living persons, however, have

shown that this is not the case (Hayek): two muscular loops remain unaffected by the cleft palate: the posterior part of the sphincter of the palatopharyngeus and the constrictor pharyngis superior. Now, these two muscles cause a narrowing of the whole oral pharynx in the direction from rear to front (Passavant) as well as from either side toward the middle (Hayek). Although the muscles of the palatum and the pharynx are not always uniformly developed, everybody who has seen cases of cleft palate can describe cases where a distinct protuberance in the middle of the posterior pharyngeal wall, and a still more pronounced lateral constriction could be observed accompanying the movements of phonation and deglutition.

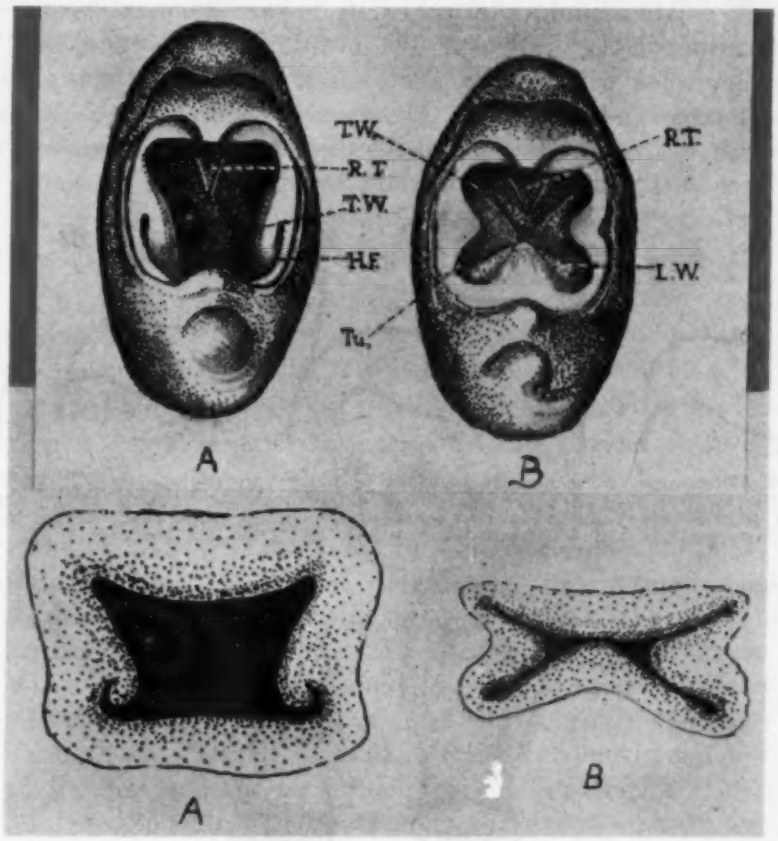


Fig. 8.—Hayek's two original photographs and their diagraphical tracing: *A*, during quiet respiration; *B*, during the maximum action in deglutition. *R.T.*, tonsilla pharyngea; *H.F.*, plica hamata; *T.W.*, torus tubarius; *Tu.*, tuba auditiva, ostium pharyngeale; *L.W.*, torus levatoris.

All the muscles described—except the azygoi—are either U-shaped or sphincters. Thus, being circular muscles, they occlude the various routes by which air or food could enter the pharynx.

PHYSIOLOGIC

Since cleft palate cases present themselves with functional disturbances, it seems appropriate at this place to deal in the following part with the nature of these disturbances.

During the acts of normal breathing, speaking, chewing, and swallowing, the muscles will always be in one of the three positions shown, changing quick-

ly from one to the other, and causing a perfectly airtight occlusion. In cases of cleft velum the different physiologic activities in the oral pharynx are considerably modified.

A. The *respiration* is impaired in one position only: breathing through the nose with open mouth, an occlusion of the oral cavity against the nasal cavity being impossible.

B. In *sucking* the position is according to III (Fig. 9), i.e., the tongue with the soft palate occludes the oral cavity toward the nasal cavity, causing a negative pressure in the oral cavity: respiration through the nose. This cannot be accomplished with a cleft palate, as the muscles of the palate cannot touch the tongue and a negative pressure, therefore, cannot be created in the oral cavity. (By the way, of all so-called suckling obturators, the aspirator according to Brophy has proved most satisfactory.)



Fig. 9.—Diagrams of the normal positions of the velum and tongue: *I*, All three routes are open—the air can pass from the pharynx into the mouth or nose. *II*, The velum raised—the cavum nasi and cavum nasopharyngeum are occluded from oral cavity and pharynx. *III*, The cavum orale is occluded from cavum nasale and from the pharynx.

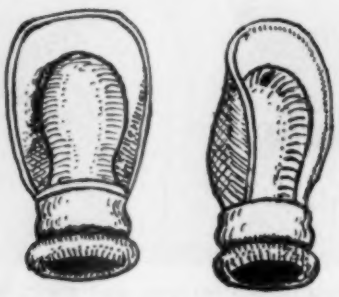


Fig. 10.—Suckling obturator according to Brophy.

C. In *mastication* the intactness of the hard palate generally is of minor importance.

D. The *deglutition,* however, is primarily started through voluntary movements of the tongue, the hard and the soft palate. According to Landois, the following separate movements can be discerned:

1. The mouth is closed by the M. orbicularis oris (N. facialis).

2. The jaws are pressed together by the masticating muscles (N. trigeminus).

3. The tip, the dorsum, and the root of the tongue, in this order, are pressed against the hard palate, pushing the contents of the mouth (bite or draught) toward the pharynx.

4. When the bolus has passed the arcus palatoglossus, its return into the oral cavity is cut off by the action of the musculi palatoglossi which stretch the anterior arches tightly toward each other and toward the raised dorsum of the tongue.

5. Now the bolus is behind the anterior arches and the root of the tongue, inside the upper part of the pharynx, where it is subjected to the successive action of the three constrictores pharyngis, being pushed past these. The action of the superior constrictor is always combined with a horizontal raising (M. levator veli palati, N. facialis) and tension (M. tensor veli palati, N. trigeminus, ganglion oticum) of the soft palate. The superior constrictor presses (through the M. pterygopharyngeus) the posterior and lateral pharyngeal wall—by causing it to bulge out—firmly against the posterior margin of the horizontally lifted and extended velum, while the borders of the posterior arches are approaching each other (M. palatopharyngeus). Thus, the cavum pharyngonasale is being occluded, rendering it impossible for the bolus to get pushed upward into the nasal cavities.

6. Dodging the contractions of the three constrictores pharyngis and of the esophageal muscles, the bolus is being pushed downward.

In applying the relevant items to the congenital cleavages of the palate, the following possibilities arise in stage 3: The tongue is pressed against the intact parts of the palate, that is to say, if the cleavage is in the center, the tongue is pressed against the lateral parts of the palate and the alveolus, obturating the cleavage. There exists, however, also the possibility that the bolus, through a movement of the tongue, e.g., by rolling it backward, is immediately being pushed toward the posterior arches. For stage 4 it must be said that the stretching of the anterior arches toward each other is out of the question in cases of cleft soft palate; instead, the dorsum or the basis, respectively, of the tongue, is raised higher, and thus the deficiency is being closed. The next move is a compensation of the incomplete action of the M. levator veli palatini and M. tensor veli palatini through a correspondingly increased contraction of the M. constrictor pharyngeus superior, especially of the M. pterygopharyngeus, viz., its most essential part. However, the mere retraction of the tongue would help to occlude the cavum pharyngonasale.

Thus, the tongue and the M. constrictor pharyngis superior are used to a greater extent in all cases of cleft palate and compensate for the lack of certain functions in the physiologically normal deglutition process. Suersen points out (in a lecture in 1867) "the incorrect substitute movements to which the tongue gets accustomed in cases of congenital deficiencies," and speaks of the necessity of correcting the habit of "these faulty movements, as soon as the tectum palati can be closed through muscular action, and a normal pronunciation is aimed at." These substitute movements of the tongue are, naturally, made also in the process of deglutition and there is no doubt that, as soon as an obturator meets the requirements, the deglutition becomes, at least partly, normal.

E. The *speech* of people with cleft palate shows a number of departures from the norm which, apart from certain abnormal functions of the pharynx, can be divided into two large groups: (1) those that are caused through the wrong functioning of the organs of articulation, and (2) those that are caused through air entering the nose.

1. *Disturbances of articulation.*—We differentiate between sounds that are created through rhythmical vibrations of the vocal cords, and those that are formed without these vibrations. The latter are merely noises, that is, irregular vibrations of the air. They are caused through a twofold process—first, air is being expelled from the lungs to which, second, obstacles are intentionally formed in the vent. It is the task of the tongue, the lips, and the soft palate to form these obstacles. We call this activity "articulation" and we speak of different zones of articulation, according to the region where the air meets the obstacle. The *first* zone of articulation lies either between the lips, or between the lower lip and the upper incisors; the *second* zone of articulation is between the tip of the tongue and the alveolus of the upper incisors, or between the tip of the tongue and the anterior part of the hard palate; the *third* zone is between the posterior part of the dorsum of the tongue and the soft palate. It is clear that a cleft palate causes only the sounds of the third zone of articulation to be pronounced incorrectly. Naturally, disturbances in the first and second zones may also occur, but these have nothing to do with the cleft palate as such.

In the third zone of articulation the explosive sounds *k* (kick) and *g* (gag) are formed, as the posterior part of the dorsum of the tongue presses against the lower part of the soft palate; then this closure is resolved with an explosive sound of air escaping. This contact being impossible with a cleft palate (the palate not being there) such patients replace these sounds with explosions caused through closure of the glottic; the sounds thus produced of course, do not resemble the *g* or *k* in the least. Similarly the composite sounds *qu* (quick) and the frictional sound *kh* (in foreign words like sheikh) belong to this group.

The sounds *l* and *r* have an exceptional position. In the English language the *l* is produced by turning the tip of the tongue slightly backward, touching the posterior border of the hard palate. The air passes at the right and left between the tongue and the velum palati or the cheeks, respectively, and leaves the mouth in a bifurcated stream at the corners of the lips, accompanied by vibrations of the vocal cords. The *r*-sounds are formed by occluding the vent at a certain place, this occlusion being opened by the escaping air, closing again immediately, being reopened several times, thus making the sound more or less burring, longer or shorter. In this context, only the palatinal *r* and the uvular *r* are of interest. The palatinal *r* is formed between the dorsum of the tongue and the soft palate with repeated interruptions of the occlusion, the uvular *r* in a similar manner between dorsum of tongue and uvula (as in Ghazi).

2. *The Rhinolalia* (pathologic entry of air into the nose or occlusion of the nasal air).—We differentiate between the closed nasal speech (rhinolalia clausa),

as, e.g., when people suffer from cold or from adenoids, and the open nasal speech (rhinolalia aperta) which is nearly always caused by cleft palate—except in cases of paralysis of the velum palati as, e.g., a consequence of diphtheria. Accumulation of the causes for the closed and the open nasal speech results in the mixed nasal speech (rhinolalia mixta), a phenomenon of great importance as far as its treatment is concerned. While the rhinolalia clausa hardly impairs the intelligibility of the speech, patients suffering from rhinolalia aperta can be understood only with difficulty. The proper sonance of many sounds is modified, while others cannot be produced at all. Proliferations and swellings in the nose, though, have a somewhat mitigating effect upon the open nasal speech, but with uranoschism their maximum result is rhinolalia mixta.

Through the raising of the velum and protrusion of the pharyngeal wall (the posterior wall according to Passavant, the lateral wall according to Hayek, see Fig. 8), a complete or almost complete occlusion between the oral and nasal cavities is caused physiologically in all sounds, with the sole exception of *m* and *n*. Such an occlusion is naturally impossible in cases of cleft palate, and the air escapes into the nose with every sound, causing the phenomenon called rhinolalia aperta, the name, however, giving us no explanation at all for this speech defect. Till 1928 it was unknown why the nose produces sounds (especially vowels) so differently than the mouth. Only comparative phonographical analysis, considering "resonance" and "reflexion," have clarified this problem.

In this context I should like to recall briefly the meaning of resonance and reflexion. If waves coming from one medium reach another medium of different density, they are thrown back into the former medium. The direction of the reflected wave depends upon the direction from which it has met the reflecting medium. Now, if a wave is being reflected from a medium of greater density, it may happen that it meets part of another wave coming from the original direction; then they may neutralize each other, for at a place where the wave should have its maximum volume, the reflected wave may arrive with its minimum volume, or vice versa (interference). "Resonance" means the intensification of sound through its being transferred from one vibrating body to another body, the "resonator" vibrating in the same rhythm as the first body. The significance of the resonance for the human voice has been pointed out by Helmholtz in his theory of resonance, which Stumpf has lately confirmed and expanded. Stumpf has proved that some of the harmonic partial tones, which naturally depend upon the pitch of the fundamental tone, are of particular importance (if, e.g., the fundamental tone is *c*, then the first partial tone is c^1, while, if g^1 is the fundamental tone, c^1 cannot possibly be an "overtone," for it is lower than g^1). These "overtones"—Stumpf calls them "main formants"—or at least their contiguous sounds in the diatonic scale, at a different pitch for each vowel, must be present in every vowel. Apart from the main formants, there exist certain lower tones—of nearly similar quality—which are important as well in producing the vowels, but which are more variable in *u* and *i*, that is, they can be replaced by still lower tones within the range of one and a half

octaves (secondary formants). Thus, if a fundamental tone is so highly pitched that its main and secondary formants cannot any more be produced, the vowels become unrecognizable, their differences blurred. The perfect vowel, that is, the vowel free from noise and vocal faults, must, therefore, contain—besides the fundamental tone (produced in the labium vocale)—a more or less substantial number of overtones, their number depending on the kind of vowel and on the pitch of the fundamental tone. In nasal sounds the unharmonic overtones are greater in numbers, and the even-numbered partial tones (as, e.g., octaves, etc.) are missing. How and where are these overtones created? Apart from the labium vocale—not to mention the whistled sounds which do not concern us here—the human vocal organ does not possess any means of creating sounds. Therefore, the overtones can be created through resonance only.

The modifications in the shapes of the resonators in the human vocal organ —as far as their shape is changeable—as, e.g., the palatinal arc, the muscles of the larynx, the soft palate, etc., do not only enable them to resonate different sounds, but influence also the conditions of reflexion, while the rigid resonators cannot contribute to the propagation of sound. Only the adaptable resonators can divert the sound, that is, the compound of basic and overtones as it exists in their area, in different directions. Therefore, it is not to be disregarded whether the soft palate form a high arc or a slight curve, as the direction in which the sound waves are reflected depends upon the direction from which they meet the obstacle.

Fröschels has shown that, in cases of cleft palate, certain rigid resonators which normally do not physiologically contribute to the formation of sound, that is, the nasal passages, are responsible for the differences in sound from the normal speech. Fröschels was struck by the similarity between a mouth with a cleft palate and one with an abnormally high palatal vault, but could never observe nasal speech in the latter cases. Therefore, he tried to plug the nasal passage with cotton wool in cases of uranoschisma. The rhinolalia aperta was thereby reduced, but not eliminated. Only when cotton wool drenched in paraffin oil was used, thus prohibiting even the penetration of the minimum of air which came through the pores of the cotton wool, the rhinolalia aperta disappeared completely. It may be imagined that, through the occlusion of the nasal passage a high Gothic palate had been created out of the intercommunicating oral and nasal cavities of the patient suffering from cleft palate, and thus the rhinolalia aperta was eliminated while respiration through the nose was not restricted. Immediately the vowels, especially *u* and *i*, sounded different, the rhinolalia aperta disappeared and sometimes even rhinolalia clausa was observed. Thus it was proved that the resonator "nasal passage" was actually responsible for the rhinolalia aperta. In the course of subsequent experiments, e.g., occlusion of the nasal passage of patients whose velum palati was paralyzed through diphtheria, and making obturators on the basis of these experiments, we came to the conclusion that the vowels *u* and *i* had found resonators in the superior nasal meatus, in the cupola of the nasal cavity, or even in the tuba, after elimination of which both vowels could be pronounced almost perfectly. The inferior nasal meatus is practically excluded as a resonator, being nearly

always closed, or nearly closed, through the turbinated bone which is usually swollen through excessive strain, in cases of cleft palate. It was proved as well, that the complete occlusion of the oral against the nasal cavity (physiologically through the velum palati) is not required—as was the classic conception—for the practical elimination of the rhinolalia aperta in cases of cleft palate.

The diagnosis of rhinolalia aperta is generally not very difficult. It is, however, not always easy to determine the degree of the disturbance and to judge the lingual performance. An experienced observer will be able to do this simply by listening, but the man with little practice will fail, because the speech of the patient with cleft palate is abnormal in various respects. These deviations from the norm are partly caused (*vide* subdisturbances of articulation) through the pathologic entry of air into the nose, while others are caused through the abnormal functioning of the organs of articulation, especially of the tongue.

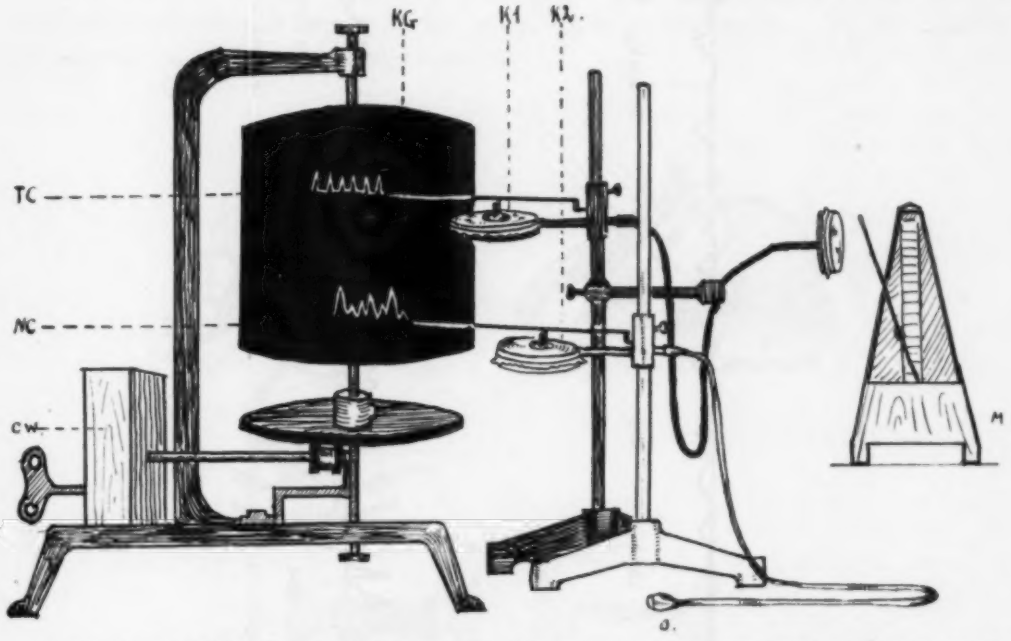


Fig. 11.—Testing the nasal blasts: *K1*, Marey-ian capsule connected with metronome /M/; *K2*, Marey-ian capsule connected with glass olive /O/—; *KG*, Kymographion; *CW*, Clockwork; *TC*, time curve; *NC*, nasal blast curve.

Unless the observer is accustomed to discerning the components in the complex of speech, he may only get the general impression of "bad speech" without being able to analyze how this impression is caused in each instance. Therefore, the inexperienced will be unable to say what exactly is wrong with the patient's speech: whether it is the fact that air enters the nose, or whether the articulation is not normal. That is why a method by which only the entry of air into the nose is registered had to be evolved. The simplest way of examining this is to place a mirror in front of the patient's nose during phonation; if the mirror gets dull, this is an indication for rhinolalia aperta. Naturally, only the graphic methods of examination are completely reliable.

For such an examination a glass olive of sufficiently wide bore is inserted in one nostril and connected by a rubber tube with a Marey-ian capsule which

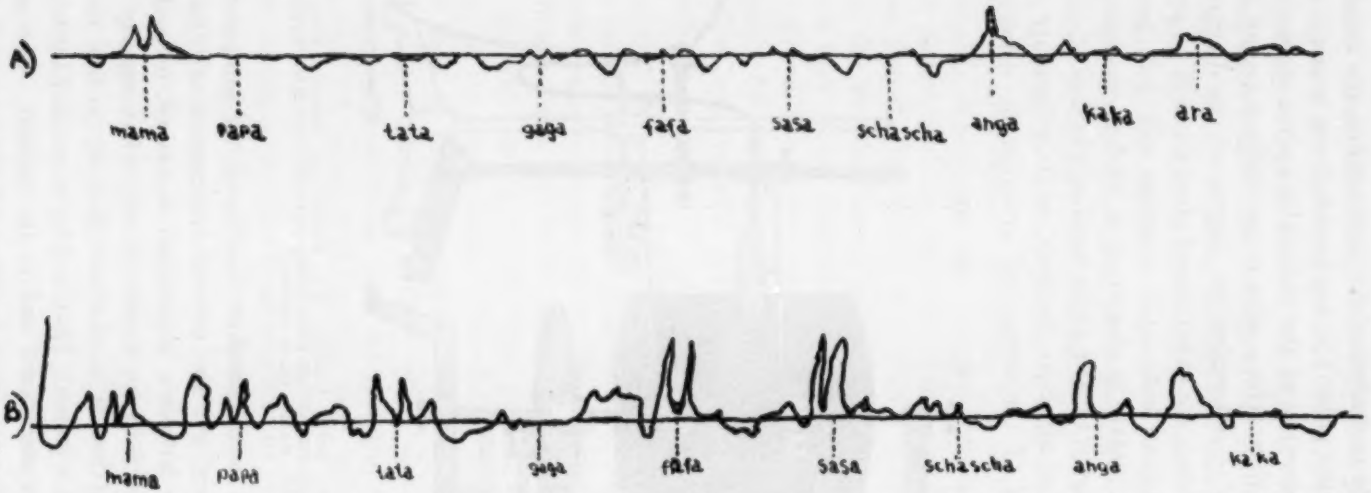


Fig. 12.—Nasal blast curves, registered by means of the Marey-ian capsule: *A*, normal patient; *B*, patient with cleft palate.

registers a curve on the kymographion covered with sooted paper. In this manner the nasal blows can be clearly recognized by comparing the normal vacillations of the writing instrument in registering the nasal sounds *n* and *m* with its pathologic vacillations when other sounds are pronounced.

The same methods of examination are used for the diagnosis of rhinolalia mixta: in such cases the patient is examined after treatment of the interior of his nose with Suprarenin, in order to reduce the swelling, and the examination is repeated later without this preparation. Thus, the part played by the rhinolalia clausa can be eliminated, and the share of the rhinolalia aperta in the disturbed speech becomes evident. This is of utmost importance for the subsequent treatment.

OBTURATORS

There are four main types of obturators: (1) the artificial vela; (2) the surgical-prosthetic methods; (3) the so-called rigid obturators; (4) the meatus obturators (nasal passage obturators).

Fig. 13.

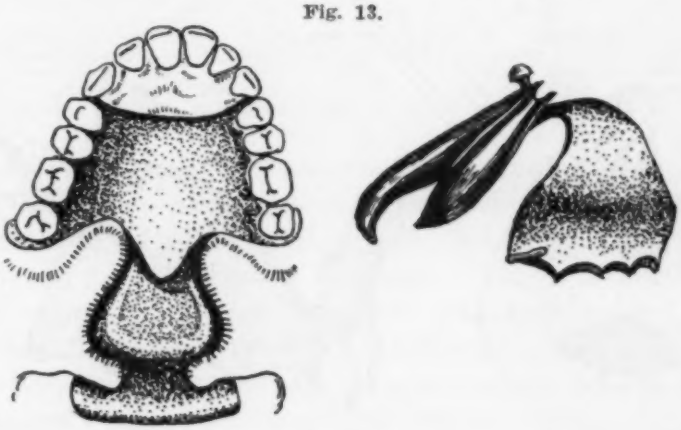




Fig. 14.

Fig. 13.—Artificial velum according to Kingsley.

Fig. 14.—Diagram of the contraction of the muscles of the soft palate causing withdrawal of the borders of the cleavage from the artificial velum (Pickerill).

1. *The Artificial Vela.*—(Snell, 1823; Kingsley, 1864; Elfert, 1914; Sörup, 1928; Claude Martin; Calvin Case.) The artificial velum according to Kingsley, as an example, consists of two plates of soft rubber, the larger of which was located above the rudiments of the velum, the smaller one beneath it, with a groove for the borders of the soft palate. These plates were rigidly fixed one

under the other, connected by a hinge with the palatinal plate. The artificial vela nowadays are hardly any more in use. The reason for their disuse is that they are based on the assumption that the borders of the cleavage approach each other during phonation, as otherwise the artificial vela could not be lifted. In certain cases this is true, but normally the cleavage is widened toward the rear during phonation (see Fig. 7).

We have shown previously that the M. levatores veli palati raise the velum and draw it backward. The result is that the borders diverge and the artificial velum loses contact.

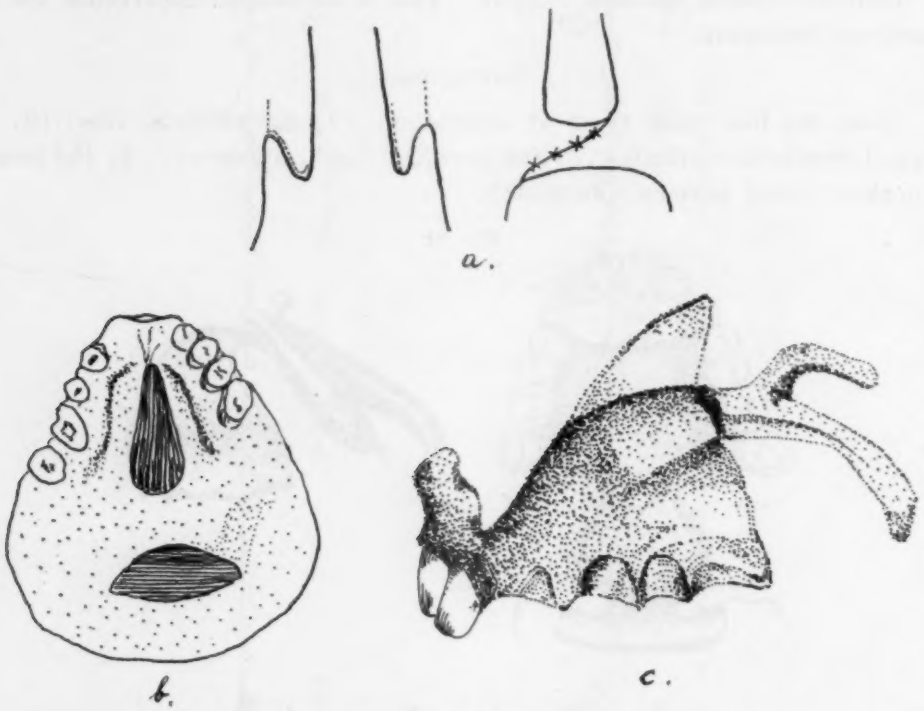


Fig. 15.—Diagram of the operation for the construction of a muscular bridge according to Pickerill: *a*, operation for muscular bridge; *b*, plaster model; *c*, an obturator (velum) according to Pickerill.

2. *The Surgical-Prosthetic Methods.*—a. Pickerill (1912) freshens the borders of the rudiments of the velum and forms a muscular bridge through suture. An artificial velum fitted with a hook which rests on that muscular bridge is connected by a hinge with a palatinal plate. During phonation the muscular bridge lifts the artificial velum, thus bringing about the occlusion of the oral and nasal cavities. After a certain period, however, the soft parts become so flaccid that their movements do not suffice any more for complete occlusion.

b. Another surgical-prosthetic method was evolved by H. D. Gillies together with Kelsey Fry. The prosthetic occlusion consists mainly in a palatinal plate, the purpose of which is to establish the greatest possible degree of mobility of the soft palate, after having joined it together, by pressing it back-

ward. This method does without surgical occlusion of the cleavage in the hard palate, which is later closed with an ordinary plate.

With both methods moderate success has been attained during the first weeks. Both, however, cause flaccidity in the soft parts (in Gillies' method at an earlier stage) to such a degree that their movements become too weak to cause an occlusion. The speech was not improved at all, and so both methods have been abandoned wherever they have been employed.



Fig. 16.—Diagram of Gillies' method with prosthetic apparatus according to Fry: *1*, direction of incision at the border between hard and soft palates; *2*, elongation of the soft palate; *3*, apparatus for putting the epithelial flaps into contact with the anterior margin of the wound in the soft palate; *4*, Fry's apparatus for elongation of the soft palate after healing; *5*, final stage—the cleavage in the hard palate is covered by a plate.

3. *The So-called "Rigid" Obturators.*—It was Suersen's idea (1866) to let the muscular bulge of the pharyngeal wall (Passavant), which protrudes during phonation, strike lightly against a rigid body, and so to cause the temporary occlusion required for the formation of sounds. That is to say, he makes use only of the movements of the pharyngeal wall (Passavant, Hayek), and neglects those of the velum, but the levatores while functioning remain in constant contact with the lateral planes of the body. To Suersen also belongs the credit for employing Schrott's method of using black gutta-percha to obtain impressions through muscular action. *Suersen's obturator* has approximately the form of a raised velum and does not occlude completely when the pharyngeal muscles are in repose. Only the protrusion of the pharyngeal muscles should suffice to create the airtight occlusion required for the pronunciation of consonants.

In cases of cleavages that affect only the posterior portion of the soft palate, and in surgical cases, a construction first described by Schiltsky was used. The original *Schiltsky obturator* consisted of a palatinal plate with a wire spiral which bore at its pharyngeal end an elastic rubber balloon, the air content of which could be replaced.

Finally, Warnekros has shown that both height and depth of Suersen's obturator could be considerably reduced, as the mass need occlude only that part of the cleavage which is left after complete contraction of the constrictor pharyngis superior. Warnekros also constructed Schiltsky's obturator for short vela rigidly from hard rubber. In cases where the rudiments of the velum palati are sufficiently movable, Warnekros fits his obturators with a hinge or a spiral spring, in order to make the maximum use of this mobility (retrograde).

Technical construction of rigid obturators: A piece of cotton wool soaked in paraffin oil is inserted into the deficiency proper. A plaster impression is taken. The cast model shows the deficiency in the hard and also partly in the soft palate. This deficiency is filled (in the model) with plaster, till a normal palatinal arch is formed. A palatinal plate is made as usual, with the proper clasps, at the same time replacing missing teeth, if any. Either a wire loop or a tailpiece, that had been premodeled in the mouth with wax, is vulcanized into the posterior end of the plate. The loop or the tailpiece, which had been roughened first, is now covered with black gutta-percha formed approximately like the raised velum, so that the pharyngeal and palatinal muscles can touch it while phonating. The compound is softened so as not to present any considerable obstacle to the functional impression of the soft parts. Thus, all the muscles concerned, when in action, automatically leave their imprint in the plastic compound.

Fig. 17.

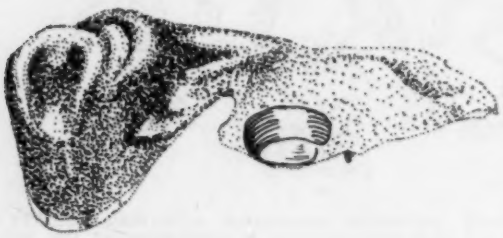




Fig. 18.

Fig. 19.

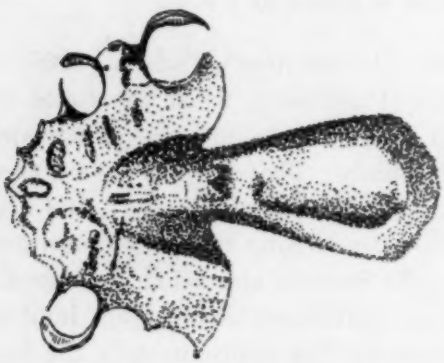




Fig. 21.



Fig. 20.

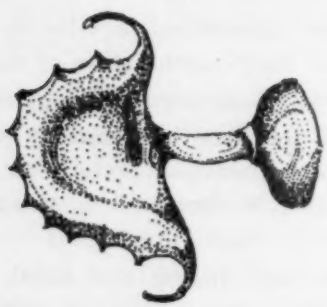


Fig. 17.—Obturator according to Suersen for open cleavages. (Observe the impressions of the tuba auditiva.)

Fig. 18.—Obturator according to Schiltsky made of soft rubber with spiral springs for operated and shortened vela.

Fig. 19.—Obturator according to Warnekros for open cleavages (modification of Suersen's obturator.)

Fig. 20.—Obturator according to Warnekros for operated and shortened vela, made of hard rubber with fixed connection by gold straps. (Modification of Schiltsky's obturator.)

Fig. 21.—Movable obturator according to Warnekros with hinge for open cleavages.

It is essential that the patient at this stage begin methodical phonetic exercises, which ought to be supervised by a specialist for disturbances of speech and voice, or by a trained teacher of phonetics. Furthermore, the patient must be instructed to remove the plastic compound obturator before meals, as otherwise a wrong picture is obtained, for the movements of deglutition are stronger than those of phonation. During the following days the obturator must be examined daily, surplus must be removed, and places showing no muscular imprints must be filled up. These alterations must be continued until the imprints of the various muscles are clearly discernible. The posterior surface of the plastic compound will then show a groove corresponding to the Passavantian bulge; the lateral surfaces must diverge from medial below to lateral above. Naturally, the upper surface does not show any impressions. It is best to have the undersurface which is facing the mouth at the level of the two halves of the velum palati as they are while phonating. If the basis is too low, nausea and vomiting reflexes are caused; if it is too high, it sometimes impedes the contact of the tongue when pronouncing *g* and *k*.

In the functional examination it may be found that the nasal sounds have not got a free passage through the nose any more; this is the case when the obturator is too bulky. A rhinolalia clausa has taken the place of the rhinolalia aperta. If, at the beginning, the nasal blasta for the consonants are nearly of equal strength, this should not be surprising. As soon as the patient, through phonetical exercises, has learned to use his muscles in the proper manner, the differences will become more marked with or without the obturator. Generally, the obturating mass will have got its proper shape in the course of two or three weeks, and the gutta-percha can then be replaced by hard rubber or plastic material.

The successes with rigid obturators as regards the function of speech depend on so many factors that they cannot be put on a general basis. There are cases where perfectly normal speech has been attained after treatment, but there are also cases of complete failure. If the rigid obturator is flawlessly constructed, the reason for this failure is that the phonetic education after insertion of the rigid obturator has not been sufficiently systematic. Only the phonetic exercises can effect a strengthening of the whole muscular apparatus, thus enabling the individual muscles to take up gradually their normal functions. This is why it is possible to reduce the size of rigid obturators after a number of years. *The dental surgeon creates only the conditions which form the basis for the work of the phonetics specialist.* The latter again is dependent upon the collaboration of the patient. Suersen has already pointed out that "after correction of the physical conditions it depends on the intelligence and perseverance of the patient, how soon and to what extent his pronunciation becomes normal." A high percentage of failures is due to an atrophy of the muscles of the posterior pharyngeal wall which renders the formation of the Passavantian bulge impossible. Then, of course, even the best rigid obturator cannot influence the amount of air escaping through the nose. However, even such patients should at least be able to learn to articulate correctly with the obturator. In such cases injections of paraffin into the posterior pharyngeal wall have been recommended, so as to create a kind of artificial Passavantian bulge. The mobility of the muscular apparatus, however, would not be improved by these injections, and the paraffin might be dislodged through the contraction of the adjoining muscles, and would then become useless. Therefore, this method has been abandoned.

Probably the most unfavorable results have been obtained after unsuccessful operations, when through tension due to cicatricial shrinking or through lack of material the velum was tightened like a drum, thus being rendered incapable of any movement. The palate, even if most beautifully stitched together, is useless if it cannot move. In such desperate cases I cut the velum surgically and make the obturator only after healing.

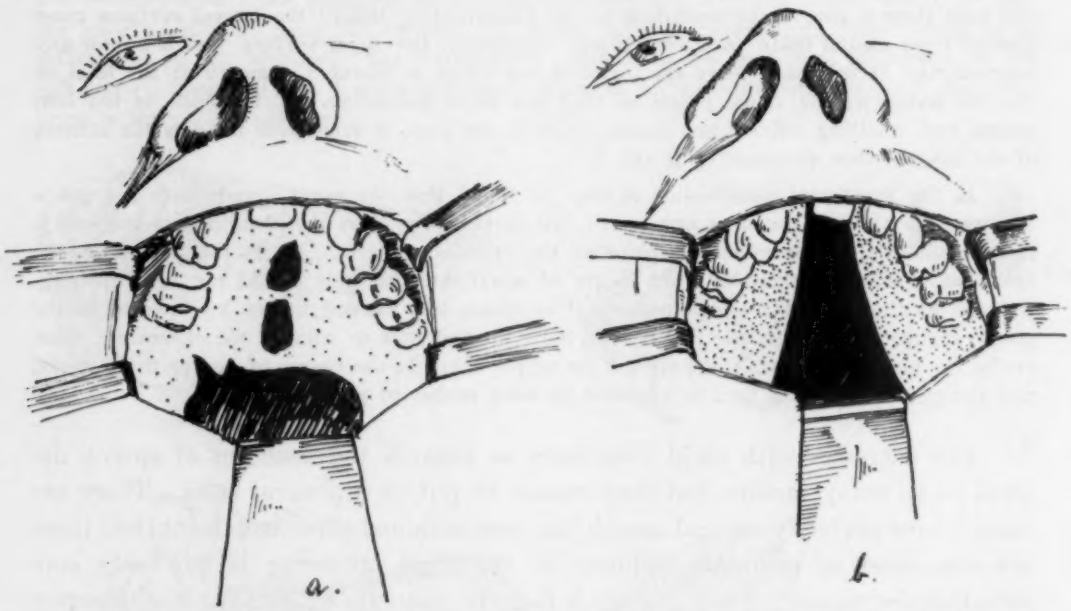


Fig. 22.—Unsuccessful closure of a cleavage in the soft palate. *a*, The velum is tightly stretched, the cleavage is traversed by several epithelial bridges. *b*, The bridges have been severed in preparation for the insertion of an obturator.

4. *The Meatus Obturators by Fröschels and Schalit.*—As previously mentioned, these obturators are based upon the presumption that in cases of cleft palate the occlusion of the cavum pharyngonasale against the cavum pharyngoorale is of no particular importance for the speech. Their construction was inspired by Fröschel's cotton wool experiment which, likewise, has already been described, that is, they occlude the meatus from the pharyngeal direction and thus prohibit the air from escaping into the nose, which would cause the rhinolalai aperta.

In the two main nasal cavities, i.e., the right and left nasal cavities which are separated from each other by the septum nasi, the following walls and apertures are discerned:

1. The roof; it is formed by the undersurface of the lamina cribrosa which is covered with mucous membrane, and by the os sphenoidale.

2. The floor; it is formed by the cranial, that is nasal, surface of the horizontal part of the os palatinum, covered with mucous membrane, and the nasal surface of the soft palate.

3. The medial wall is formed by the septum.

4. The anterior wall corresponds to the roof of the anterior prominent part.

5. The posterior wall in its upper regions is formed by the anterior wall of the sphenoid bone; it is open below, taken up by the choana.

6. The lateral wall; the three conchae (superior, media, and inferior) together with the lateral and lower nasal bone are forming the three nasal meatuses (superior, medius, and inferior). Medially the nasal meatuses open into one cavity which extends at both sides of the septum toward the rear, and which is called meatus nasi communis. Toward the pharynx the lateral wall of the nasal cavity ends in a fold, the plica nasopharyngea.

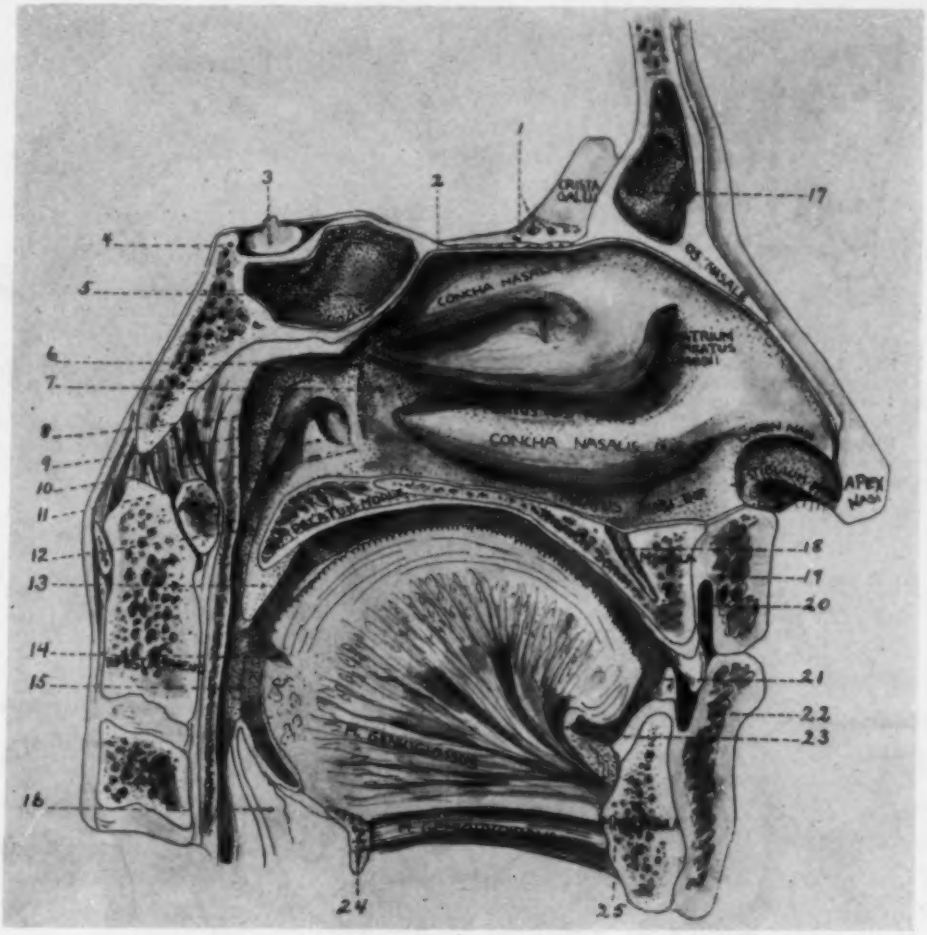


Fig. 23.—Sagittal cut through the facial part of the head and the three upper vertebrae of the neck. The cut was made to the right of the medial axis; the septum has been removed (according to Rauber-Kopsch); *1*, Lamina cribrosa. *2*, Recessus sphenoethmoidalis. *3*, Hypophysis. *4*, Dorsum sellae. *5*, Septum sinuum sphenoidalium. *6*, Arcus choanae. *7*, Meatus nasopharyngeus. *8*, Recessus pharyngeus (Rosenmülleri). *9*, Torus tubarius. *10*, Torus levatoris. *11*, Labium anterius. *12*, Plica salpingopharyngea. *13*, Uvula (palatina). *14*, Foramen caecum (Morgagnii). *15*, Plica palatopharyngea. *16*, Epiglottis. *17*, Sinus frontalis. *18*, Canalis incisivus. *19*, Labium superius. *20*, Vestibulum oris. *21*, Cavum oris proprium. *22*, Labium inferius. *23*, Tunica mucosa sublingualis. *24*, Os hyoideum. *25*, Spina mentalis.

7. The anterior openings for the nostrils, the nares.

8. The posterior openings, the choanae, are formed by the posterior border of the partes horizontales ossi palatini below, by the posterior border of the vomer medially, by the undersurface of the sphenoid above, and by the medial lamella of the processus pterygoideus laterally. When performing a rhinoscopia posterior (see Fig. 26), the dividing wall between the choanae (vomer), the posterior end of the inferior concha, more or less hidden the posterior end of the middle concha, and still less clearly the superior concha can be observed, depending on the position of the palatum molle.

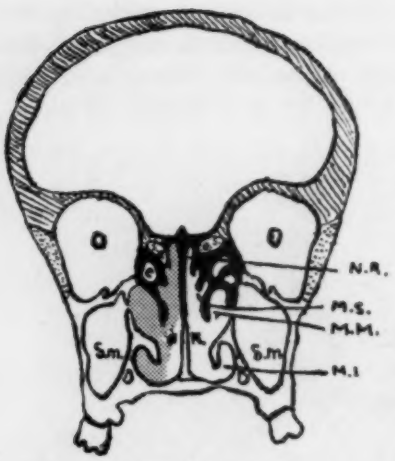


Fig. 24.



Fig. 25.

Fig. 24.—Frontal cut through the head according to Corning: *S.M.*, sinus maxillaris; *S.e.*, cellulae ethmoidales; *N.*, cavum nasi; *O*, orbita; *N.R.*, nasal roof; *M.S.*, meatus nasi superior; *M.M.*, meatus nasi medius; *M.i.*, meatus nasi inferior.

In the left part of the picture, the meatuses are dotted to illustrate Fröschels' experiment.

Fig. 25.—Plaster cast of the right nasal cavity, seen from the right: *1*, meatus superior; *2*, meatus medius; *3*, meatus inferior; *4*, apertura nasi externa; *5*, recessus pharyngeus (Rosenmülleri); *6*, ostium pharyngeum tubae auditivae. (According to Rauber-Kopsch.)

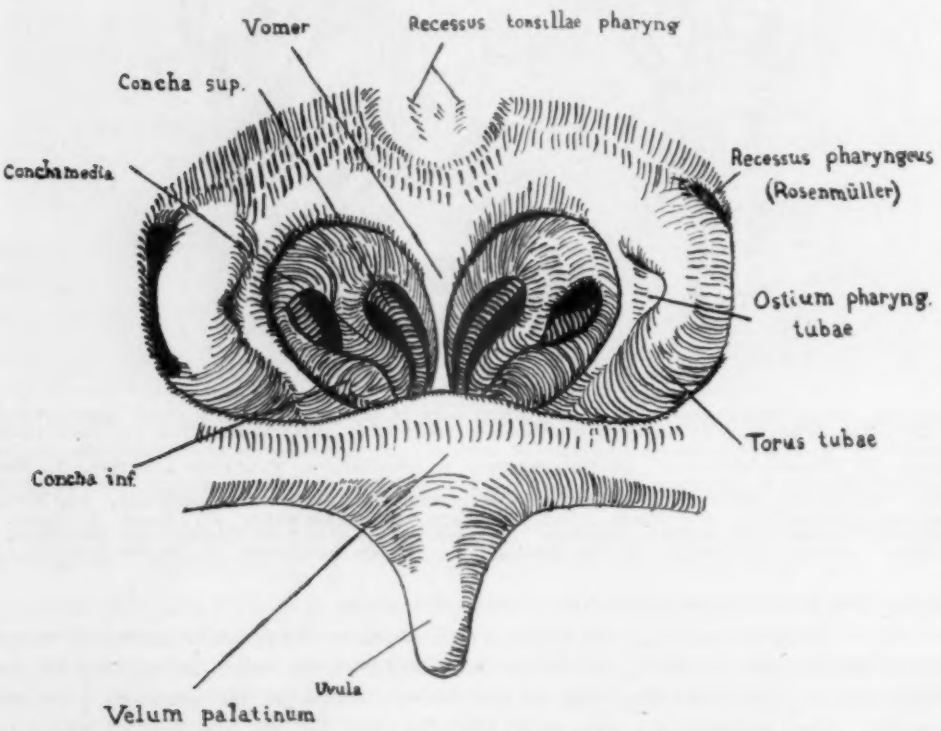


Fig. 26.—View of the choanae in rhinoscopia posterior. (According to Corning.)

What are the conditions in cases of cleft palate? Through the lack, or wide cleavage, of the two palatinal plates—except a bilateral complete cleavage—the cavum pharyngonasale communicates with the cavum pharyngo-orale, thus creating quite abnormal conditions. The superior, anterior, and posterior walls are the same as in normal cases, except that the choana does not exist.

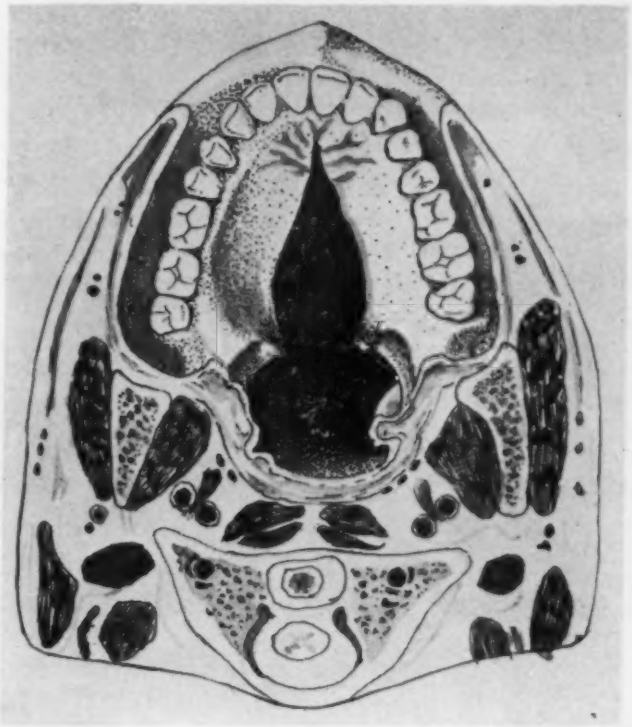


Fig. 27.—Horizontal cut through the skull of a case of cleft palate. (According to Ernst.)

The medial wall of the nasal cavity, the septum nasi in its cartilagenous as well as in its bony part, is in most cases shortened, its free border protruding into the common oral-nasal cavity. Besides, the septum may be malformed, the vomer may be bent or lengthened in its total extent. The lateral wall of the nasal cavity evidently suffers changes only secondarily through its unnatural intercommunication with the mouth: through the pathologic strain the conchae, especially the inferior concha, become swollen and secondarily indurated. Thus, the inferior meatus in most cases is completely occluded, and the medial and superior meati are narrower than normal. The meatus nasi communis is practically nonexistent, as it forms one cavity with the cavum pharyngo-orale. The inferior wall is composed, as in normal cases, by the nasal surface of the partes horizontales ossi palatini and the nasal surface of the soft palate, but only partly, viz., laterally. Medially, the inferior wall shows the pathologic deficiency of the uranoschisma. The anterior apertures are of no importance in this context. The posterior apertures, the choanae, are open below and medially, thus giving a clear view of all three conchae and their meatuses in the rhinoscopia posterior, even with an ordinary oral mirror.

Fig. 28.



Fig. 29.



Fig. 30.

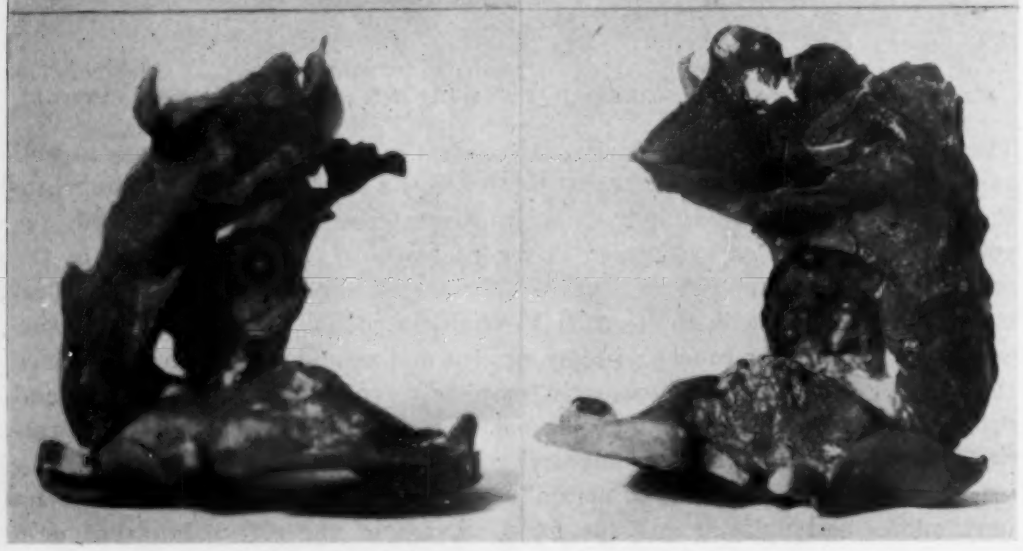


Fig. 28.—Palatinal plate with "Kerr-core."

Fig. 29.—Palatinal plate with the gutta-percha impression of the cavum nasopharyngeale.

Fig. 30.—Palatinal plate with the gutta-percha impression of the cavum nasopharyngeale. (Observe the impression of the tuba auditiva.)

The nasal meatus obturator (Fröschels-Schalit) consists of a palatinal plate and a perforated lump which is rigidly connected with the plate. This hard lump occludes the nasal passages, yet permits free nasal respiration.

After construction of a palatinal plate with a wire loop, the loop is covered with Kerr compound or with S. S. White Modelling Tray Compound in such a manner that it is up to one centimeter distant from the posterior pharyngeal wall. After having ascertained—by rhinoscopia posterior—that this "core" is sufficiently removed from all surrounding walls, an ample quantity of softened black gutta-percha is added, the whole apparatus inserted into the mouth, and the patient is asked to speak loudly a few sentences. After a few minutes the first tests can be made, as to whether the nasal cavity has actually been completely occluded, that is, whether the rhinolalia aperta has become a rhinolalia clausa. It will often be found that, after the first insertion of the gutta-percha mass, the rhinolalia aperta has either not been influenced at all, or has become a rhinolalia mixta.



Fig. 31.



Fig. 32.

Fig. 31.—Lump of gutta-percha on an old obturator according to Warnekros (Schiltsky), the palate being closed and the velum considerably shortened.

Fig. 32.—Gutta-percha mass for occlusion of the nasal meatuses on an old obturator according to Warnekros (Suersen) for a case of open cleavage.

Through rhinoscopia posterior, which can be performed with an ordinary oral mirror, it can be ascertained at what places the gutta-percha has not completely plugged the aperture; more gutta-percha is added at the respective places, and the apparatus is inserted again. This must be continued till a complete rhinolalia clausa has been created, and the patient is unable, even with an effort, to press air through the nose.

In this connection I have to mention that in certain cases also the aperture of the tuba Eustachii has to be closed, i.e., in cases where the bulge of the levator is insufficiently developed. The resulting impressions, which have grotesque and fantastic shapes, must now be "shaved" and reinserted, whereupon the rhinoscopic control is repeated. Now the size of the lump has to be reduced, which is still much too bulky. After modeling the lingual wall, the lump is flattened by vertical cuttings of its frontal wall, and again controlled. Thereupon a hole is drilled through the gutta-percha lump as precisely as possible in the middle, with a conical drill, thus giving the patient enough air for breathing through both nostrils. These air holes often present difficulties, and

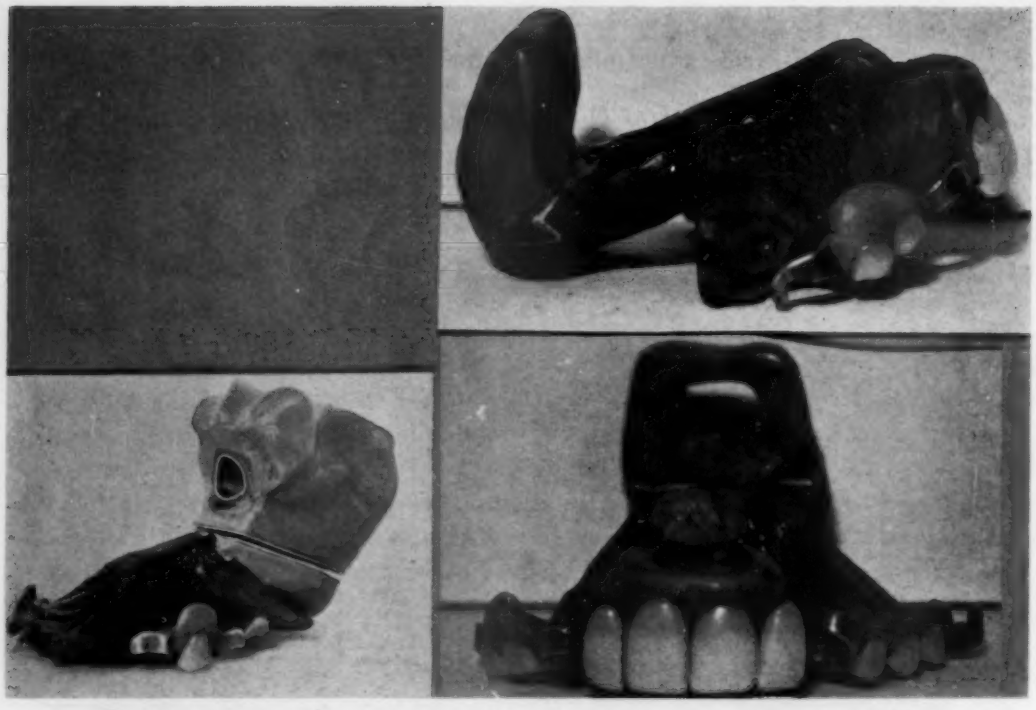


Fig. 33. Fig. 34.

Fig. 33.—The first meatus obturator according to Fröschels-Schalit for open cleft palates. (For demonstration purposes the hard rubber lump was detachable.)

Fig. 34.—Meatus obturator (Fröschels-Schalit) for open cleft palate, obturating only the pharyngeal apertures of the nasal passages. (First model, on an old obturator according to Warnekros-Suersen; for demonstration purposes the hard rubber lump was detachable.)

Fig. 35.

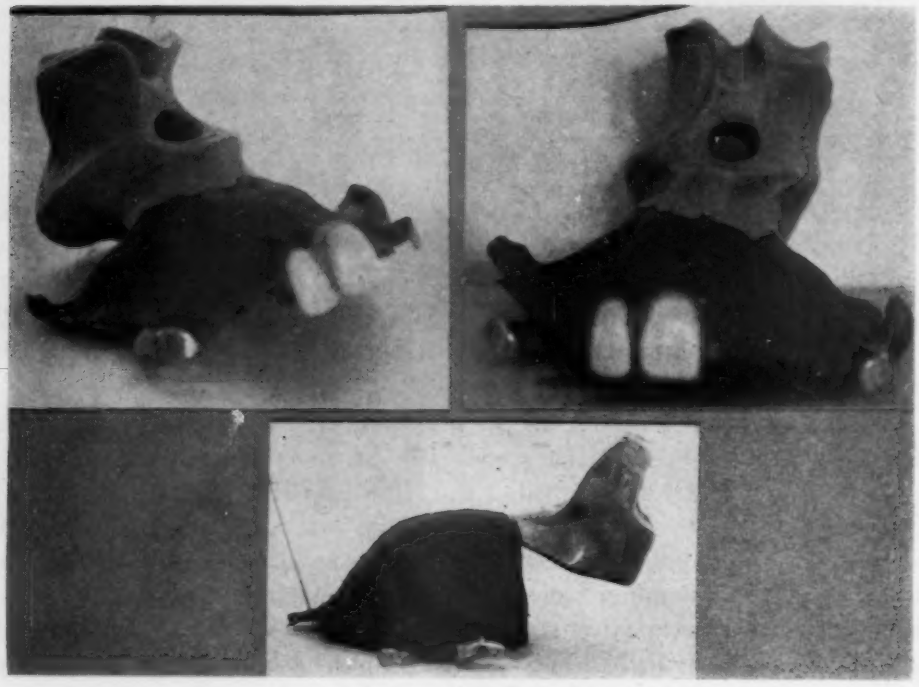


Fig. 36.

Fig. 35.—Meatus obturator (Fröschels-Schalit) for open cleavage. (Here the impressions of the considerably swollen concha inferior are easy to discern.)

Fig. 36.—Meatus obturator (Fröschels-Schalit) for a wide cleavage and high vaulted (Gothic) palate, occluding the nasal meatuses till about half their length. (From the first series of experiments.)

experiments must be repeated several times, until the most suitable place for them is found. In cases of cleavages which go through on one side in particular, it is sometimes advisable to have two separate holes (see Fig. 37). Now if everything has been accomplished, the rhinolalia aperta must have vanished completely, while the free breathing through the nose is unimpeded, and the patient must be able to speak without any trace of a twang. The further procedure is simply to replace the gutta-percha by hard rubber or by a resinous material. Instead of the many weeks it usually takes to make the impressions for a rigid obturator, the method described here requires one sitting only.

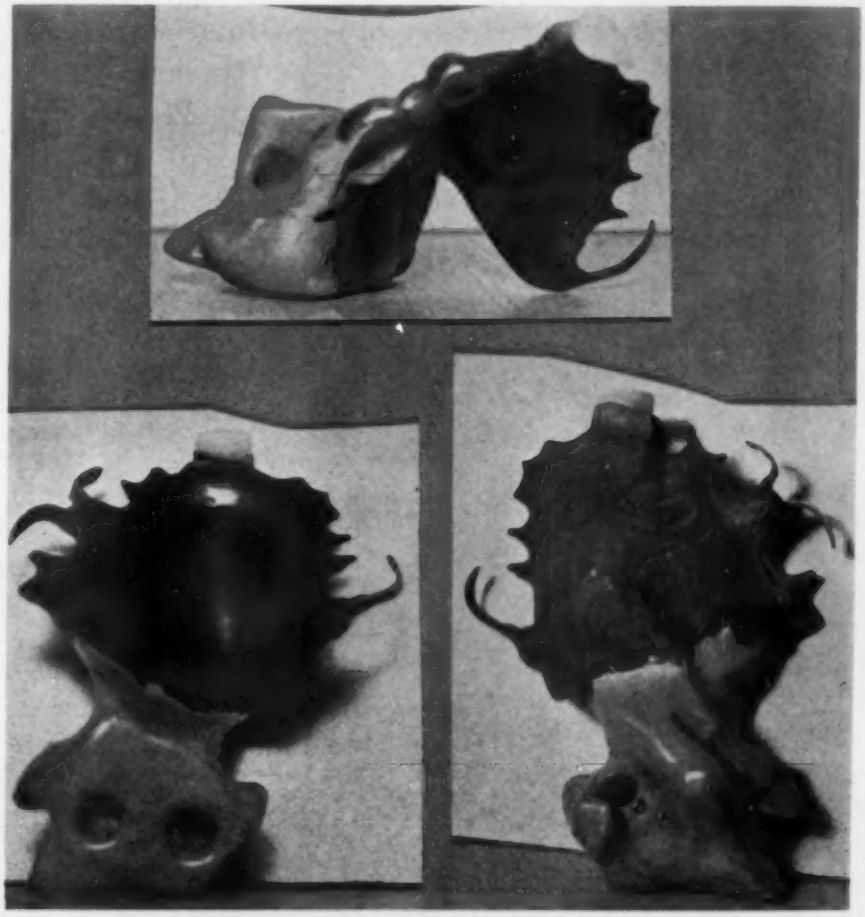


Fig. 37.—Meatus obturator (Fröschels-Schalit) for a cleavage traversing the palate on one side only. (Observe the two air holes, the impressions of the oblique septum and of the tuba auditiva to the left.)

After completion of the obturator, another examination is necessary, so as to make sure that it fits perfectly in every place, and to make adjustments, if necessary. Then the patient is sent to the logopedist for correction of his articulation.

The successes attained with the meatus obturators with regard to the function of speech, especially regarding the elimination of nasal speech, and enabling

the patient to pronounce the nasal sounds (*m* and *n*), are remarkable. Analytical sound tests have proved the superiority of the meatus obturator over all the other systems hitherto known. While the rigid obturators as such do not bring about any alteration of sound by influencing the nasal speech, but only the function of the muscles after their strengthening through exercises causing the necessary separation of the oral from the nasal cavity, it can be clearly seen from sound analysis that things are quite different with meatus obturators: already the occluding apparatus causes an improvement of sound. *At once, and without any exercises, the rhinolalia aperta disappears completely,* and only the disturbances of articulation remain. These latter disturbances are, however, as already repeatedly stressed, only to be cured by conscientious treatment by a logopedist or a teacher of phonetics.

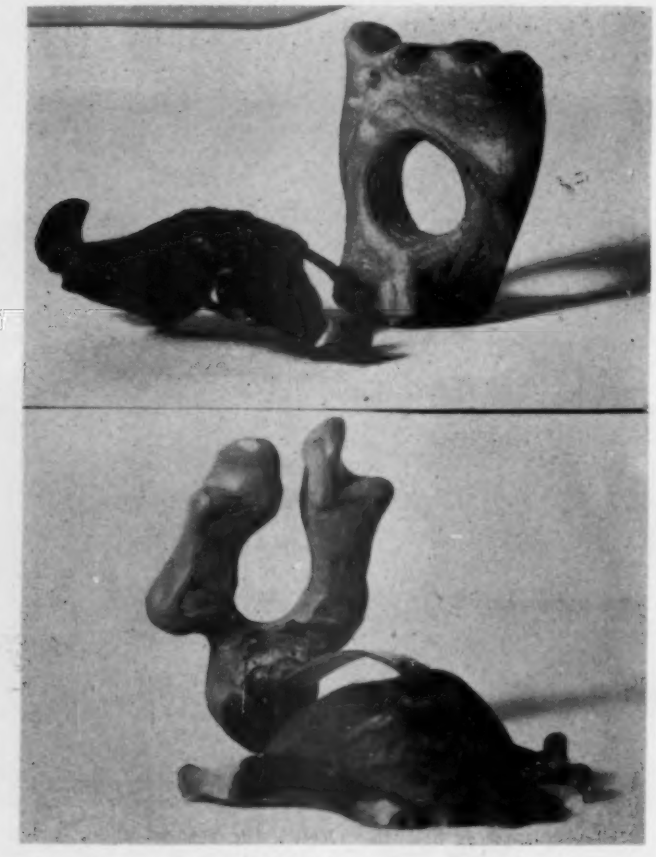


Fig. 38.—Meatus obturators (Fröschels-Schalit) for a staphyloraphically closed palate and shortened velum.

Finally, the question arises, which obturator should be used for which cases? As already mentioned in the beginning, obturators are indicated only in cases which cannot be operated upon, or which have already been operated unsuccessfully. The most frequent form of nonsurgical cases is the uranocoloboma posticum, as complete cleavages can usually be operated by surgical closing of the hard palate. On the basis of the given anatomic status, two sub-

divisions can be distinguished: (a) cases with strongly developed pharyngeal muscles, and (b) cases with underdeveloped pharyngeal muscles.

a. In cases of strongly developed pharyngeal muscles, in which the vestibulum pharyngis can be sufficiently contracted in spite of the existing cleavage, and which justify the expectation that they can be operated upon at a later date, or that after thorough treatment with exercises the obturator can be done without, a rigid obturator according to Warnekros is indicated.

b. In cases of little-developed pharyngeal muscles, an obturator according to Fröschels-Schalit is indicated.

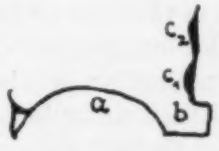


Fig. 39.

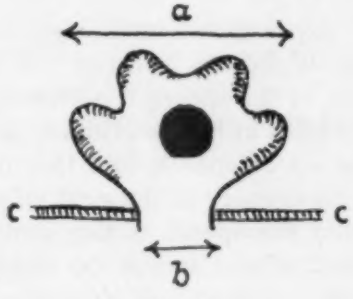


Fig. 40.

Fig. 39.—Diagram of a meatus obturator which would have to circumvene the lower concha which is considerably enlarged in a pharyngeal direction. *a*, Palatinal plate; *b*, space for the pharyngeal end of the hypertrophical inferior concha; *c*, pharyngeal occlusion of the medial and superior meatuses.

Fig. 40.—Diagram of a meatus obturator for a very narrow cleavage. Its upper width (*a*) is greater than the width of the existing cleavage (*b*).

After unsuccessful operation, and for the improvement of the results of an operation, it is the question whether the cleavage is situated in the hard palate (then a simple occluding button is sufficient), or whether the cleavage extends into the soft palate. In the latter case the meatus obturator is the method of choice, except in such cases where the insertion of the obturator is impossible for anatomic reasons. In cases of very pronounced hypertrophia of the concha, when it extends very far pharyngeally, the meatus obturator would have to be shaped like a bayonet, that is, it could not be inserted.

In cases of very narrow cleavages, where the rudiments of the velum are very limp and reach very far back, which would not allow for the passage of the lump to the pharyngeal wall, this lump, of course, cannot be inserted through the narrow cleavage.

In such cases a meatus obturator would not be practical and a rigid obturator according to Warnekros must do.

During twenty years' practice as a surgeon of the jaw in Europe and in the Near East, the author had the opportunities of seeing more than a thousand cases of cleft palates, and to make obturators in nearly four hundred cases. On the basis of this experience this article has been written, and it may be concluded with the quotation: "For I will restore health unto thee, and I will heal thee of thy wounds, saith the Lord, because they called thee an outcast. . . ." (Jeremiah 30:17.)

THE RESPONSIBILITY OF THE SPEECH CORRECTIONIST IN THE TREATMENT OF THE CLEFT PALATE PATIENT WHO HAS RECEIVED SURGICAL OR PROSTHETIC TREATMENT

HERBERT KOEPP-BAKER, M.S., PH.D.,* CHICAGO, ILL.

UNIVERSAL experience convincingly attests to the primacy of speech in the totality of human behavior. A fact less commonly appreciated is that the integrity of the speech function figures importantly as a determinant of psychosocial health of the individual. It is in the light of these two facts—that of the primacy of speech, and that of the integrity of the speech function—that the full urgency of the cleft palate patient's needs become manifest. Since the capacity for speech is the most distinguishing human attribute, it follows that what affects speech favorably or unfavorably affects the most human part of us.

A somatic derangement as extensive as cleft palate will obviously be advertised in a profound functional disturbance. Functional evidences of aberrant organic states assume significance in terms of the level upon which they operate. Some variations of structure have only limited importance because they affect adversely functions that are singularly personal and individualistic. An illustration is the absence of an eye or a leg. Here, locomotion or sensory perception are interfered with. These functions are of primary importance to the person, but their social significance is secondary. Speech, on the other hand, is indisputably a social function. Hence, any disturbance in its integrity resulting from atypic structure assumes additional significance because of its adverse effect upon social adjustment. Its personal importance, though not incidental, is clearly subordinate.

It is evident then why the speech clinician conceives himself to be engaged by the *core* of the problem of rehabilitating the cleft palate patient. He reasons that the full personal and social impact of the disorder is registered through the patient's inadequate speech. Rightly or wrongly, he argues that the success of any and all remedial procedures must be measured in terms of improved speech. He is supported in this observation by the patient's own testimony, for it is unusual indeed to find a patient who does not give first place to his hope for a recovery of speech. Implicit in the adoption of this view is the acceptance of serious clinical responsibilities.

Despite the general recognition of the centrality of the speech needs of the cleft palate patient, a number of practical factors militate against their easy, rapid, and certain satisfaction. First, it is apparent that the patient's speech improvement is, in a large measure, predetermined by the success of

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prosthesis or surgery. It is not without significance, therefore, that under the scheme of clinical service that usually obtains, the speech clinician enters late into the coordinated effort to rehabilitate the patient. Further, his results are conditioned by a complex of factors over which he has little or no control: such as, the intelligence and competence of the prosthesis or surgery, the native intellectual and emotional endowments of his patient, and, finally, the fact that the changes he seeks to achieve are more tenuous than the rearrangement of tissue or the construction of a mechanical device.

The speech clinician who is sensitive to his clinical responsibilities will be frank to admit that he is not at all content with the present clinical dividends of speech treatment. His major obligation to the patient and to his profession lies in his persistent effort to discover and to remedy the inefficiency of the service his own specialty agrees to provide. But he believes, too, that there is mutual advantage to be derived from an open, frank, and searching discussion of the present inadequate clinical services to the cleft palate patient.

It is consonant with such a frank inquiry to observe that there is a predetermined margin of disadvantage in the fact that the speech re-education generally, though incorrectly, is presumed to *follow* prosthesis or surgery. Speech clinicians have come meekly to inherit an attitude that they must, as a matter of course, accept and be content with what they get from prosthodontia or surgery. This is unfortunate for the patient. It is wholly defensible to assume that the speech correctionist's work should both precede and be concurrent with the other specialistic services to the patient. Some very practical results would accrue by having the speech clinician's influence felt earlier rather than later in the sequence of clinical events.

It is disheartening for the patient and often discouraging to the prosthetist and the surgeon for the patient to discover that the outcome he seeks most eagerly, namely, improvement of speech, does not immediately or inevitably result from the fitting of a speech aid or from a surgical operation. Often this disappointment is for both the clinician and the patient a deterrent to further effective effort. Therefore, speech clinicians are beginning to see the value of providing their patients with presurgical and preprosthetic speech training and psychological conditioning. Many of the patient's most disturbing speech "mal-habits" are correctable before the prosthodontist or the surgeon begins or completes his work. Such psychological and speech training greatly increases the patient's insight into the extent and character of the difficulties inherent in speech recovery. He comes to see by this means that prosthesis and surgery *are but steps* of preparation for something that he must do for himself. In the case of children the effect of such antecedent speech training is especially marked.

The speech problem in the cleft palate patient is actually tripartite. It involves the improvement of intelligibility, the correction of nasality, and the establishment of mental hygiene. Of these, the attack upon the problem of intelligibility may well precede everything else. There is ample evidence that intelligibility can be greatly improved without surgery or prosthesis. To

do so is to provide immediate encouragement for the patient and reduce much of the bewilderment and frustration so characteristic of the problem.

But there are even more pressing reasons why the speech correctionist should feel responsible for and participant in the early clinical decisions. The study of the speech of the patient and his total personality, as well as his reactions to systematic training, will provide important facts that should affect positively the complex decision as to whether prosthesis or surgery, or both, should be employed. The present practice of looking into a child's or adult's mouth in a single examination and determining what should be done for the speech mechanism upon the meager data so derived is, frankly, leaving much to intuition and little to fact. If the speech clinician comes into the joint consultation early and well prepared, he will be able to guide properly a decision that he knows to be critical because it is so final.

Regardless of how successful prosthesis or surgery may be, the patient still has a learning task of great proportions. The speech clinician cannot too often or too earnestly remind the prosthetist and the surgeon of the great individual differences in the capacity to learn speech. Without a doubt, nonunions of the midline of the mouth produce the most disordered of all speech patterns. No matter how effective prosthesis or surgery are, the patient must still reorder his speech with an essentially imperfect mechanism. Therefore, a realistic appraisal of the patient's intellectual and emotional capacity to benefit by clinical procedures is a basic requisite for a sound prognosis. Without such careful and inclusive estimates of the patient's intellectual and physical abilities and limitations, we are under great handicap in trying to compare scientifically the results of various methods of treatment. The type of service provided by the speech clinician lends itself to a complete evaluation of the personality in whom the fissured mouth is but one factor of importance.

Thoughtful and conscientious specialists are fast becoming aware that unless a sound and balanced cooperation among surgeons, prosthetists, and speech correctionists can be achieved, the clinical rewards in the treatment of the cleft palate patient will continue to be disappointing. Functional cooperation, however, demands an integrating goal. Experience is making it evident that the most effective clinical team can be developed only when speech improvement becomes the primary end sought and the criterion by which every clinical procedure is evaluated.

In such a clinical service-structure the speech correctionist assumes his proper place and stature, and his true function becomes apparent. His point of view, training, and experience can be most effectively applied in such a setting. The speech correctionist presumes to know the nature of the speech function and the requirements for its improvement. He recognizes these as being multiple. They are anatomic, physiologic, phonetic, and psychologic. With a proper blend of scientific humility and self-confidence he should stand ready to apply the most searching criticism not only to his own procedures of rehabilitation, but also to those of the prosthetist and the surgeon. He is prepared to suggest ways to improve the treatment of cleft palate patients. These suggestions need not be concerned with or confined exclusively to his

own techniques. His recommendations in collateral areas may be accepted in good faith if he reflects through them intelligence, insight, freedom from prejudice, and adequate understanding of the basic facts of the other specialties. The character of the central problem in restitution of the cleft palate patient is clearly defined. It is the improvement of the speech and, through it, of personality for effective living. This leaves no doubt of the nature of the clinical responsibilities of the speech clinician.

PRESENT STATUS OF SPEECH TRAINING OF PATIENTS WHO WEAR SPEECH CORRECTION APPLIANCES

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UNFORTUNATELY, the literature on speech pathology and correction contains almost no information on the speech training of cleft palate patients who wear prosthetic speech aids. Even one of the most recently published books, devoted entirely to the cleft palate patient, contains only two general references to an obturator. This may give the impression to the uninformed that speech correction methods for obturator cases are identical with those used for cases with surgical repair. Both types of patients exhibit similar speech patterns, both before and after surgical or prosthetic intervention, but each presents problems of training peculiar to itself.

It would be difficult to report comprehensively upon the status of speech training of prosthetic cases throughout the country, without benefit of a survey. It is necessary, therefore, to limit this paper to a brief statement, within the knowledge of the writer, of the extent of the work being done in speech training of cleft palate patients who wear prosthetic speech aids; and to a description of methods, procedures, and policies which are employed in a few localities in Pennsylvania. We are aware of the fact that some work in this area of speech correction is being done at the University of Michigan, State University of Iowa, University of Denver, and The Joseph Samuels Dental Clinic, Rhode Island Hospital. We have no information concerning the exact nature of the work being done in these centers. It is to be hoped that publication of the proceedings of this meeting will motivate workers in other centers to increase their activities in this field, and to share, through publication, their observations and findings which might prove beneficial to others in the profession.

In the remainder of this paper we shall attempt to give an account of the programs of speech training for cleft palate patients who wear prosthetic appliances as reported by the following clinical centers: Pennsylvania State College, Lancaster Rotary Club Cleft Palate Speech Clinic, School of Dentistry, University of Pittsburgh, The Speech Clinic of the Psychological Clinic and The Surgical Prosthesis Clinic of the Evans Institute of Dentistry, at the University of Pennsylvania. The methods, policies, facilities, equipment, and personnel vary among these centers. The needs of patients with similar difficulties also vary. Speech correction always requires highly individualized planning for successful results. In this brief report, we shall be able to present only a general composite picture of the methods and procedures now being used in these centers.

Read before the Academy of Cleft Palate Prosthesis, April 10, 1946.

*In charge of The Speech Clinic of the Psychological Clinic, University of Pennsylvania.

The speech correctionist should participate in the diagnosis and initial planning of a program for the rehabilitation of a cleft palate case. Consultation by the prosthodontist, oral surgeon, orthodontist, and speech correctionist make it possible to determine more effectively the steps which should be taken first, the extent to which surgery can aid, what dental regulation is possible, and the features of the appliance to be made. The speech correctionist can do his part by pointing out how best results in speech production can be obtained. Once the prosthetic program is under way, the speech correctionist can cooperate in the designing and fitting of the appliance. During this period of construction of the aid, the speech correctionist can also help to prepare the patient to accept the obturator. He should be told that it will serve as a speech aid, but that it will not assure the patient normal speech; that even the most skillfully constructed aid, effectively placed, will not as a rule result in much change in the patient's speech without speech training. He can be shown the difference between the process of speech in an individual with an intact soft palate, and one with a prosthetic replacement. Finally, the prosthetist should contribute to this preparatory program by explaining how the aid should fit, the probability of frequent adjustments at first, and should give instructions for proper care of the appliance.

Before beginning speech training of this type of patient, one should secure a measure of his intelligence, conduct an audiometric examination, and make a phonographic recording of his speech—before and after placement of the appliance. It is not always possible to make these examinations, consequently it is sometimes necessary for the examiner to estimate the intelligence of the patient by reference to his school record and by careful observation during the preliminary contacts. If one has any reason to suspect a hearing loss, he should refer the patient to an otologist for an appraisal of his auditory acuity as well as to determine the possibility of a pathological condition in the hearing mechanism. Normal hearing is essential to the development of normal speech.

Sometimes speech training can be started before the patient has been fitted with his speech aid. There is a difference of opinion over this point. At least there are a few speech skills which usually cannot be taught until the oral cavity has been made somewhat intact. For example, if a patient has been substituting a glottal stop for a *k* or *g* sound, he will not be able to correct this faulty habit until he has a palatal surface against which to press his tongue to effect this stop consonant. Also, it would be discouraging for a patient to attempt to correct the nasal quality of his speech without a means of securing a closure of the nasopharyngeal port. Training of the muscles involved in effecting this closure can begin at the time of the placement of the appliance, or even before. This training may have little meaning to the patient, however, until he sees and feels the obturator in place, and can observe its purpose. A mirror can be used to aid in this type of instruction.

The cleft palate patient who has been fitted with a prosthetic speech aid is confronted with several specific problems: (1) directing the air stream and

sound through the mouth—conversely, deflecting it from entering the nasal resonator; (2) learning how to articulate all consonant speech sounds correctly; (3) training his ear to discriminate between a correctly and incorrectly articulated sound; this involves the psychological problem of accepting the new and different sound as a desirable, intelligible one; (4) overcoming accessory compensatory reactions, such as contraction of the alae to effect a nasal closure. These habits usually persist even after speech has improved. The first three problems can be attacked simultaneously, but require careful analysis and instruction for the patient. For example, in teaching him how to make a correct *s* sound, one can explain that a stream of air passing through a grooved tongue and creating a sharp friction against the incisal edges of the upper anterior teeth is essential. It is acceptable for the patient, while learning to make the sound, to pinch his nose occasionally in order to facilitate channeling the air stream through the mouth. Most patients who wear appliances, however, will still make the *s* sound defectively. To the observer, his *s* sound will have a whirring quality, apparently caused by the friction of air against the appliance and other structures in the posterior part of the oral cavity. It is difficult to observe exactly what happens, but a patient can usually be made aware of the fact that it is a posterior mouth sound. He must be taught to become conscious of a fricative sound made at the anterior opening of the mouth. Sometimes simple experimentation with whistling or slight hissing sounds made by changing the position of lips and tongue rapidly bring the desired results. Once the patient has succeeded in making a sound which approximates a good *s* sound, he must be trained to observe how he made the sound and what auditory characteristics the sound has. After he has learned to make the sound in isolation, he can be instructed to practice it in combination with a series of vowels, such as *s-ee, s-oh, s-oo.* This helps to fix the new speech habit. From then on, the patient practices the sound in more meaningful units of words, phrases, and sentences, until the correct enunciation of the sound becomes habitual. At the beginning of training for a specific sound the patient may complain that "it doesn't sound right." What this really means is that it does not seem correct because it sounds different from the utterance to which the patient has become accustomed. He will need to have enough confidence in the correctionist to accept the new pattern as correct, and practice it. Members of his family and friends may observe the improvement in the patient's ability to make the sounds, and their comments help to convince the patient that the sound he has been practicing is an intelligible one, and is preferred to his former faulty production of the sound. After learning to make the sound in the initial position of a word, it is sometimes necessary to practice it in the medial and final positions in a variety of words.

This method of teaching the *s* sound has been described somewhat in detail as an illustration of phonetic training for the fricative consonant sounds. A cleft palate case may encounter similar difficulty in learning to articulate *th* and *f*, and their voiced equivalents. *Sh* and *tsh* (*ch*air), and their voiced equivalents, may also give some trouble. All fricative speech sounds require channeling of

the breath stream through the mouth cavity, with a certain adjustment of the articulators, resulting in the production of friction on the lips, teeth, or tongue, or a combination of two of these structures, in the front part of the mouth. Learning how to produce any speech sound correctly requires skillful auditory discrimination.

Nearly all patients with unrepaired cleft palates, and those with surgical repair who have received no speech training, substitute a glottal stop for *k* and *g*. They have learned to effect a closure of the glottis of the vocal cords in place of a tongue-palatal closure in the oral cavity, in order to approximate the characteristic plosive quality of these sounds. After a patient has been fitted with a prosthetic speech aid, he will need to be trained to make the *k* and *g* sounds properly. He is not apt to do this by auditory stimulation alone, but will require detailed instruction in proper placement and movement of the tongue.

Improving the nasal voice quality of a patient with a cleft palate is a difficult task and is dependent in large part upon the available equipment for effecting a nasopharyngeal closure. Successful surgical repair provides the patient with a means of closure which is equivalent to that employed by persons whose velar action has always been normal. He will require speech training, however, to learn how to use his improved palate effectively. In the case of a prosthetic replacement, the problem is quite different. Time does not permit an adequate discussion of this extremely important problem, but several points can be mentioned. The posterior end of the appliance, sometimes referred to as the bulb, should be of such a size and shape, and should be placed in such a position in the oral pharynx, as to permit a closure of the nasopharyngeal opening by the action of the pharyngeal and palatal muscles responsible for the sphincteric closure in this area. This statement may appear to be too obvious to be repeated here. But the clinical application of this principle is quite another matter. There is still disagreement as to the proper construction and placement of the obturator, but it appears incumbent upon both prosthodontist and speech pathologist to study this problem, and observe the results clinically of various types of construction and installation. Assuming, however, that a patient has been equipped with a mechanical aid which is adequate both as to construction and placement, there still remains the problem of training the patient how to use his muscles to procure a closure when it is required for correct utterance. Koepp-Baker describes this process as a matter of "fractionating the swallowing reflex." That is, the nasopharyngeal closure can be brought about by voluntary control of certain muscle responses involved in the act of swallowing. In some cases, however, it is possible to obtain good speech results without especial emphasis upon this type of muscle training. In spite of a good closure, however, ear training again is essential. The cleft palate patient is aware of the fact that his speech suffers from a loss of intelligibility because of the reactions of his auditors, but he does not know *how* his speech sounds to others. This poses both an auditory and a psychological problem which is not uncommon in some types of voice disorders. The

patient must learn not only how to make the correct sound, i.e., free from nasality, but also to accept it and use it habitually.

One other point should be made concerning voice training in this type of cleft palate case. As the palatal and pharyngeal muscles develop, after placement of the prosthetic speech aid, the patient may manifest a de-nasality. That is, the nasopharyngeal opening may become too small to permit a normal nasal resonance for the sounds *m*, *n*, and *ng*. This tends to convert these nasal continuant sounds into oral plosives, producing *b*, *d*, and *g*, respectively. This difficulty can be remedied by reducing the size of the bulb, but care must be taken to make this reduction in a manner which will still permit an effective closure. The surfaces to be reduced should be determined by observing the action of the muscles in relation to the obturator.

As the cleft palate patient approaches the termination of his formal speech training, the speech correctionist may then attack the problem of compensatory reactions, other than the glottal stop. These reactions occur usually in the form of various facial contortions, resulting, as pointed out previously, from an effort to prevent nasal escape of air while attempting to utter stop and fricative consonant sounds. If correction of this sort is attempted too early, it may tend to cause the patient to become self-conscious. Furthermore, the task of learning how to make new sounds is difficult enough without adding the complication of trying to eliminate specific accessory muscle habits.

These patients should receive both individual instruction and group training. A combination of the two types of teaching can usually be managed if the membership of the group is limited to six or eight persons. Every patient requires sufficient individual attention to assure his knowing how to make every speech sound correctly, otherwise his group work may serve only to encourage the persistence of his faulty habits. The prime advantage of group participation is the favorable psychological effect it produces upon the individual members. In a well-managed group of speech cases, one can observe several psychological processes which operate to help the members as individuals: (1) An emotional relationship with the instructor who serves as leader of the group. This gives the individual confidence and a feeling of security, which facilitates learning. (2) An acceptance of the purpose for which the group was organized. This tends to develop comfortable interrelationships between the members of the group. (3) A willingness to accept one's problem of defective speech, resulting in an improved personal adjustment to his handicap. This is brought about by a realization of the fact that others have a similar problem, which helps him to take an objective attitude toward his difficulty. (4) An opportunity for the individual to learn how to help himself by observing the errors and attempts at correction on the part of other members. All of these factors serve to motivate the individual to make an effort to improve his speech.

In conclusion, we would like to point out the uniqueness of cleft palate speech. The cleft palate patient is not merely manifesting deviations in voice quality and distortions in consonant articulation. It is the totality of his

speech pattern which is different. He is speaking habitually in a manner which has resulted from a congenitally imperfect mechanism. The speech correctionist, therefore, does not simply train the patient to make a sound differently, but he attempts to reorganize the total speech pattern, to make it possible for the patient to speak more normally. The rehabilitation of the cleft palate patient, therefore, requires skillful surgery, orthodontics, and prosthodontia; careful training in voice production, articulation, and auditory discrimination; and finally, psychological preparation for accepting and using a type of speech to which the patient has hitherto been unaccustomed.

A TECHNIQUE TO SPLIT TEETH

R. Y. Sung, D.D.S.,* Philadelphia, Pa.

IN ATTEMPTING to remove an impacted third molar, it is generally agreed that in some cases, the tooth should be first split and then removed sectionally. To split a tooth means to break its unity and to decrease the resistant force of being removed.

The usual way to split a tooth is to use a mallet and a chisel (or an automatic mallet) or a revolving drill. This has at least five disadvantages: (1) The tooth is not always split in the desired manner. (2) It is impossible to split a tooth after it has been loosened. (3) A mallet and a chisel produce a great deal of shocking force, and the drill produces a large amount of heat and dust which are harmful to the surrounding tissues. (4) When the mallet is handled by the assistant, it is the assistant who splits the tooth. (5) It generally requires a longer time to section a tooth with a drill.

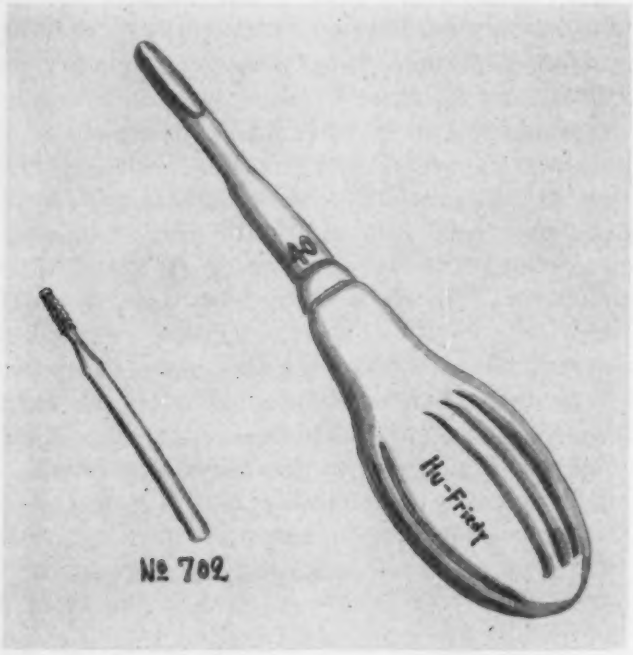


Fig. 1.—Instruments used for tooth division.

About four years ago, I worked out a technique which is so efficient that most teeth can be split within three to five minutes and it is so simple that almost anyone can use it.

*From the Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

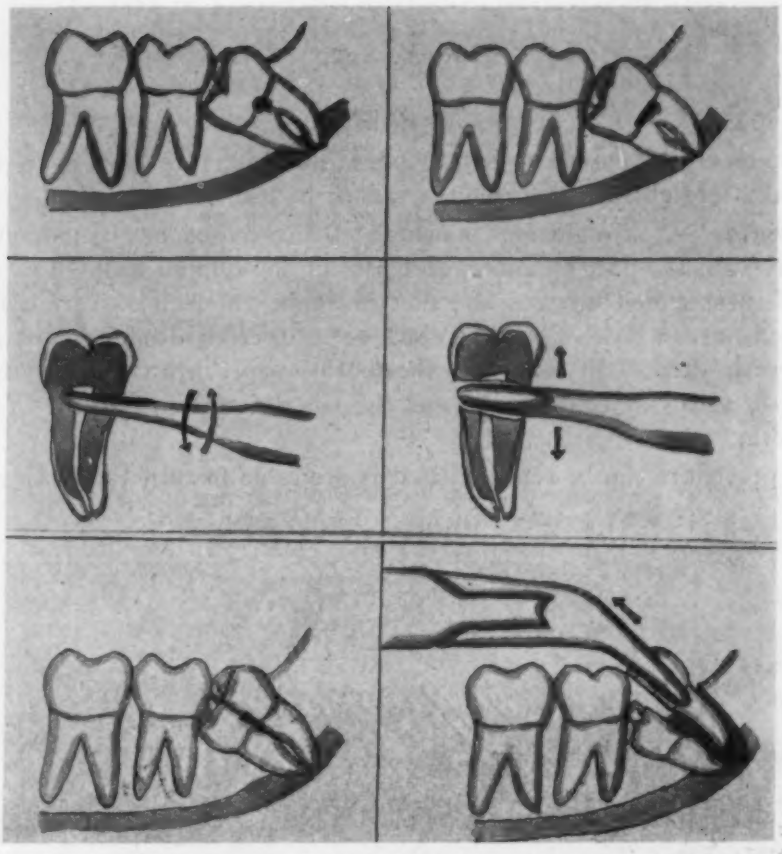


Fig. 2.

Fig. 3.

Fig. 2.—To amputate the crown first is the best way to remove impactions.

Fig. 3.—The impacted $\overline{|8}$ can be split longitudinally and have the distal section removed by an upper premolar forceps.

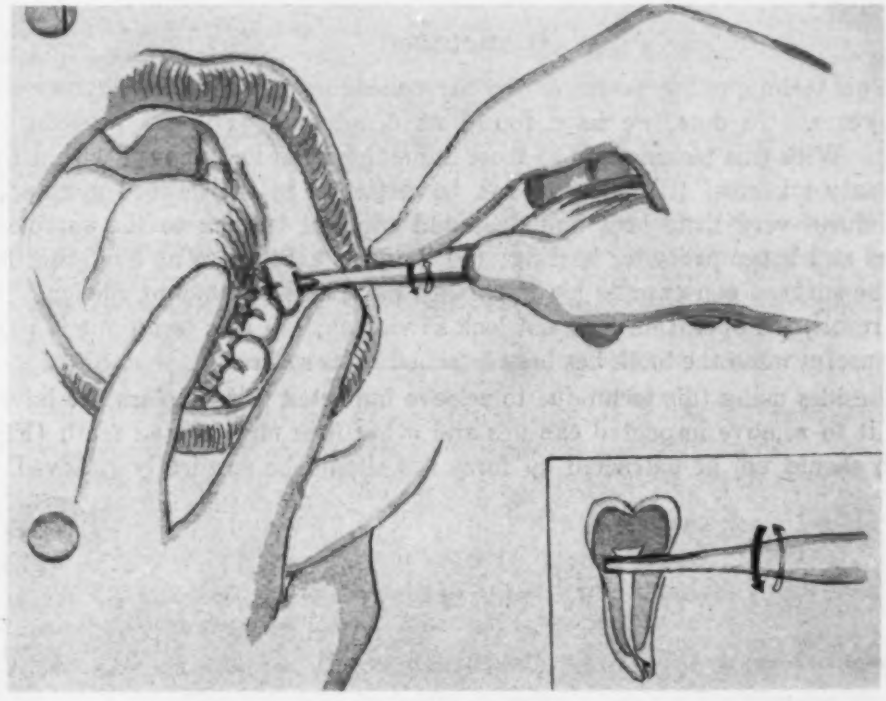


Fig. 4.—After the slit has been made, a straight elevator is inserted deep into the slit, and, with a gentle turn of the elevator, the tooth is split into two desired sections.

TECHNIQUE

Instruments.—A crosscut fissure burr, No. 702, bibeveled by a revolving stone, and a straight elevator, such as shown in Fig. 1.

Procedure.—A straight hole is made at the cementoenamel junction through the buccal wall, the pulp chamber, and part of the lingual wall. The revolving burr is then extended upward and downward in order to convert the straight hole into a narrow slit. Then the elevator is inserted deep into the slit, and, with a gentle turn of the elevator, the tooth is split into two desired sections. (Figs. 2, 3, and 4). The direction of the slit determines the line of cleavage of the tooth.

The procedure can be repeated if it is desirable to split the tooth into more sections.

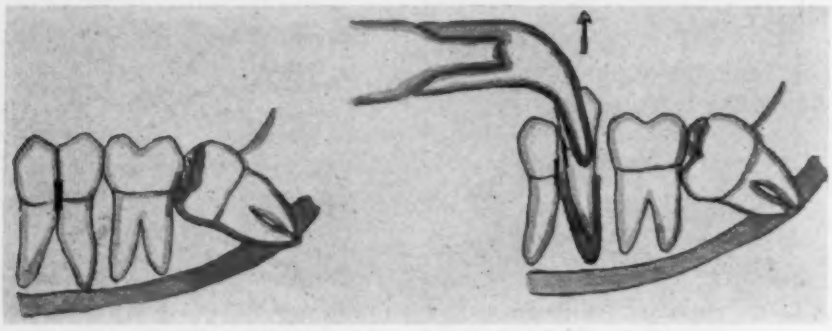


Fig. 5.—After the $\lceil\overline{6}$ has been split longitudinally, each section can be removed by a lower incisor forceps.

DISCUSSION

This technique has been used by my colleagues in China and by myself for four years. To date, we have found no disadvantages in its procedure and result. With this technique, (1) most impactions can be removed within fifteen to twenty minutes; (2) all teeth can be sectioned in the desired manner; (3) it produces very little heat and dust and surgical trauma to the surrounding tissues and hence promotes healing; (4) patients experience no malleting force; (5) the surgeon can execute his work with poise and paramount efficiency; and, as a result, the operation does not look so violent; (6) this technique is particularly useful when the tooth has been loosened in its socket.

Besides using this technique to remove impacted third molars, we have also used it to remove impacted canines and other firm multirooted teeth (Fig. 5). Teeth should not be extracted by force but should be surgically removed.

Case Report

CASE NO. 97

ATTEMPTED SUICIDE WITH GUNSHOT THROUGH THE MOUTH. UNEXPECTED LOCATION OF THE PROJECTILE

IMRE JAKABFI, M.D.,* DEBRECEN, HUNGARY

SHOTS from revolvers of appropriate caliber directed from the floor of the mouth toward the cranial base, as often seen in suicide, are usually lethal. The following case is reported in which a patient with an injury of this kind survived without considerable consequences.

The patient, a man of 21 years, was mutilated during the war; he lost his left arm. He attempted suicide by means of a shot with a revolver directed into the region of his chin. After two trial shots, he pressed the orifice of the revolver to the region below his chin and fired. Surprisingly he did not lose consciousness, but there was bleeding of some duration and he swallowed some blood. After a few hours he called for assistance and was brought to our clinic.

Below the chin, at the upper border of the hyoid bone in the median line, could be seen a gunshot wound. It was 1 cm. in diameter, of circular shape, and surrounded by a ring of soot and gunpowder granules. The surrounding tissues and the whole submental region were swollen and tender. In the base of the tongue, at a distance of 7 cm. from its tip, in the median line, a wound could be seen, which was of circular shape, 1 cm. in diameter, and covered by a grayish fur, surrounded by a hyperemic halo. A third wound was present in the soft palate, somewhat laterally from the insertion of the uvula; this was of lenticular shape and extent, with a yellowish-grayish fur, surrounded by a hemorrhagic and hyperemic halo. In the epipharynx, nasal cavity, and larynx no pathologic changes were found. The patient displayed severe aphonia, which may have been due to psychical shock as he regained his voice after he was told that his throat was uninjured.

The roentgenogram of the cranial base showed a slight impression of the clivus Blumenbachii, but no other pathologic changes. The bullet was not seen in the head; it was found in the ileocecal region, lying supposedly in the cecum.

By means of laxatives and a high enema the projectile was removed. It was of 9 mm. caliber, a lead bullet with a steel cover, and on its tip, corresponding to the impact with the bone, concentrical circular deformations could be seen.

Except for minor suppuration of the skin, the patient recovered uneventfully and was dismissed from the clinic in three days.

After penetration of the floor of the mouth, tongue, and soft palate, the living force of the bullet was not strong enough as to penetrate the massive bony resistance of the clivus. This may be due to a quantitative or qualitative insufficiency of the gunpowder. The projectile recoiled from the cranial base, impressing it slightly. Yet this impression was without any further clinical importance. Then passing the epipharynx and choanae it reached the oropharynx and was swallowed.

SUMMARY

A case has been reported which is unique in medical literature. This case gives proof of the importance of a thorough physical examination of every patient.

Publication authorized by the Oto-Rhino-Laryngological Clinic of the University in Debrecen, Hungary.

*Senior assistant of the staff of the Oto-Rhino-Laryngological Clinic, Debrecen, Hungary.

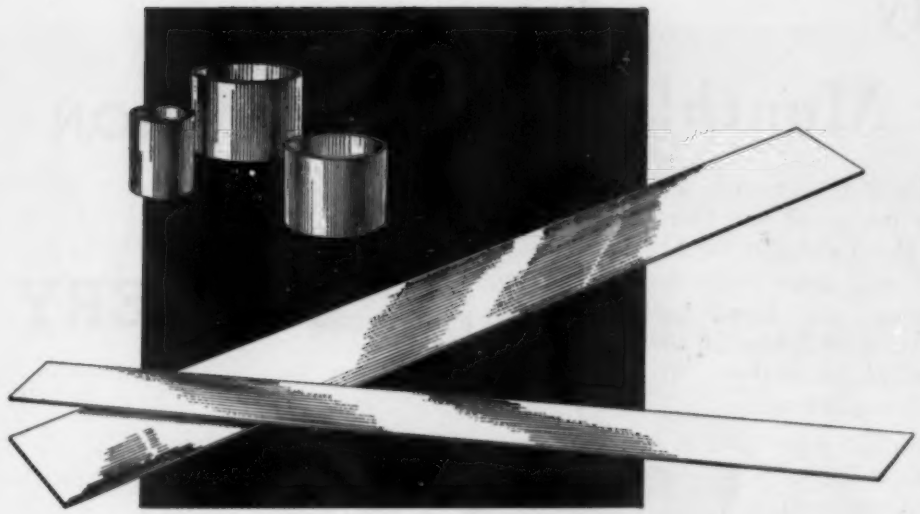
Editorial

The Care of the Cleft Palate Patient

WE ARE publishing in this issue of the JOURNAL a number of papers dealing with *the rehabilitation of the cleft palate patient.* The majority of these papers were read at the meeting of the *American Academy of Cleft Palate Prosthesis,* held in Harrisburg, Pennsylvania, on April 10, 1946. That the hare-lip and cleft palate patient presents an important public health problem has been recognized in Michigan, where all children born with such defects are cared for by the state. No doubt such an arrangement makes it possible to furnish these unfortunate patients optimum results not only because it eliminates the financial factor, which no doubt prevents many from submitting to what in most cases spells prolonged and expensive treatment, but also because such patients are then taken care of in medical centers where there are properly coordinated facilities for the highly specialized professional services, and the right nursing and general care required for such cases.

Dr. Kemper, of the University Hospital of the University of Michigan, pointed out that rehabilitation of the cleft palate patient may be likened to a great drama in which there are a number of actors each playing an important role under the direction of one individual, the surgeon, who is responsible for its management and production. He should see the patient through the various phases of the treatment until the patient is able to take his rightful place in society and carry on in a highly competitive way of life. This may take many years and will require the cooperation of specialists in many fields: the orthodontist, the prosthodontist, the speech correctionist, and the psychologist. All these will share in the responsibility of getting the optimum result by their combined efforts. The papers in this issue deal with some of these problems.

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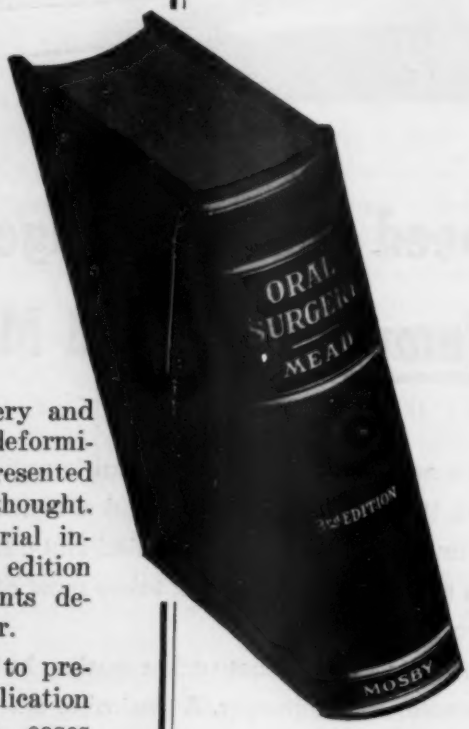
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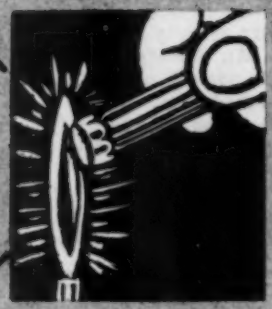
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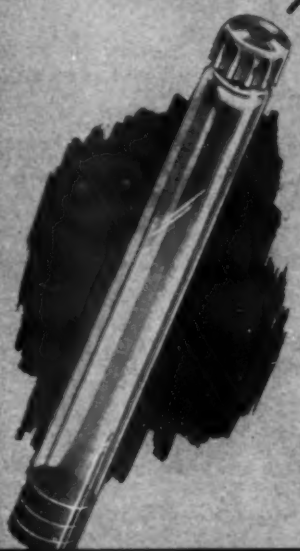
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Had just closed their eyes for a much-needed nap,
When out of the night there arose such a clatter
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"All Christmas Seal Stamps have been turned into cash!"
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A ONE, a FIVE, a TEN, a TWENTY,
Fifties and hundreds and thousands a-plenty.
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To prevent and control tuberculosis.
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To cut down the rate and the root of infections.
—Vocational counselling to guide us back
And keep us on the healthy track.
So we closed our eyes with a grateful smile,
And slept and dreamed for a little while.
And God, whose job it is to nurse
A sick and ailing universe,
Thoughtfully nodded His kindly head
Then, in His voice of mercy said,
"Blessings on this freely-given wealth,
Merry Christmas to all and to all...GOOD HEALTH."



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Original Articles

A CLINIC ON THE DIFFERENTIAL DIAGNOSIS OF ORAL LESIONS

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Diagnosis of oral lesions has been dependent for many years upon experience of the clinician. Textbooks have devoted little space to diagnosis of special oral conditions and it is relatively recently that two textbooks on oral diagnosis have been published.[1, 2] The teaching of diagnosis in dental schools is likewise one of the newer disciplines. It is true that some schools have given courses in "examination," or in diagnosis and treatment planning, but the lack of organized, well-conducted courses is emphasized by the recent discussion on diagnosis at the 1946 meeting of the American Association of Dental Schools.[3]

The use of Kodachrome reproductions has simplified the teaching of diagnosis by direct lecture, but, unfortunately, satisfactory reproduction of these color photographs is so expensive that their general use in illustrating periodical contributions and texts is precluded. The following "clinic" is presented in the original with color illustrations, but in these discussions an effort will be made to paint in the color with words.

1. LESIONS OF THE HARD TISSUES OF THE TEETH

a. *Hypoplasia of Enamel.*—Enamel hypoplasia is a condition characterized by grooves, pits, and similiar defects on the enamel surfaces and is caused by arrested development during the stage of enamel apposition. Enamel is formed in two stages: in the first, the uncalcified enamel matrix is laid down over the dentin (apposition), and in the second stage the enamel becomes calcified (calcification). Failure or arrest of the first stage results in enamel agenesis or enamel hypoplasia. Failure in the second stage results in enamel



Fig. 1.—Enamel hypoplasia in Negro boy with a calcium deficiency background. The incisal edges indicate a probably prenatal or neonatal disturbance, and the labial grooves a disturbance in enamel deposition at about the third year of life. Note also the geminated mandibular left first and second incisors and the negroid pigmentation of the gingivae.

hypocalcification. The underlying cause of enamel hypoplasia may be nutritional (calcium or vitamin D deficiency, rickets, tetany, etc.), infectious (febrile diseases, syphilis, extension of dental infection, etc.), or physical injury.



Fig. 2.—Enamel hypoplasia with holes entirely through the incisor crowns. These areas were forming between the second and third years of life.

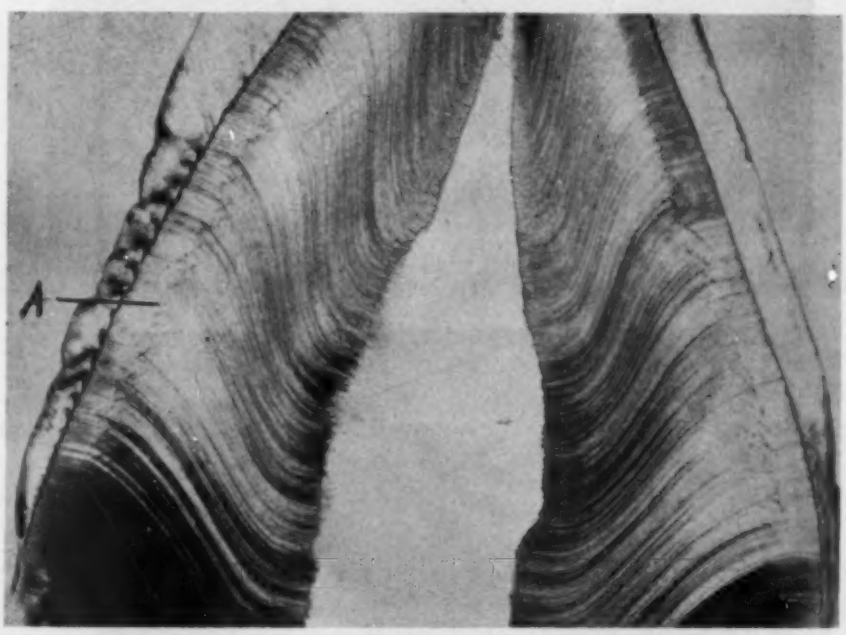


Fig. 3.—Section of hypoplastic tooth crown (ground section) showing defects in enamel and corresponding interglobular areas in dentin. (From Applebaum, E.: J. D. Res. 17: 181, 1938.)

Grossly, the teeth are characterized by grooves, pits (Fig. 1) or, in extreme cases, holes (Fig. 2). These defects are seen most prominently on the labial and buccal surfaces, although the occlusal and incisal surfaces may be severely disturbed. The region affected is that which was being laid down at the time of the disturbance. Reference to a chart, such as that of Schour and Poncher,[4] will show the normal time of development of the part that is defective. Thus, hypoplasia at the mid-buccal surface of the premolars cannot be

blamed on scarlet fever at 2 years of age, for apposition of this part did not take place until the third to fifth year of life.

Microscopically (Fig. 3), defective formation of enamel and of the underlying dentin is seen.

A variation of hypoplasia which is the result of congenital syphilis was described by Hutchinson[5] in 1856. This stigma, now known as Hutchinson's teeth, is one part of a triad, the other two of which are interstitial keratitis and deafness of eighth nerve origin. The permanent incisors, canines, and first molars which are being apposed at the time of syphilitic infection are more commonly affected. The incisors are screwdriver-shaped or pegged and commonly

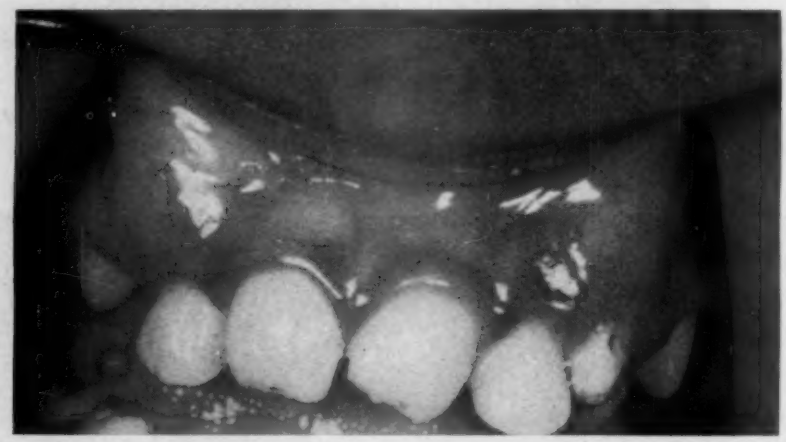


Fig. 4.—Hutchinson's incisor in Negro boy. The teeth are screwdriver-shaped with mesial and distal bulges and notched incisal edges. Note also negroid pigmentation of gingivae.

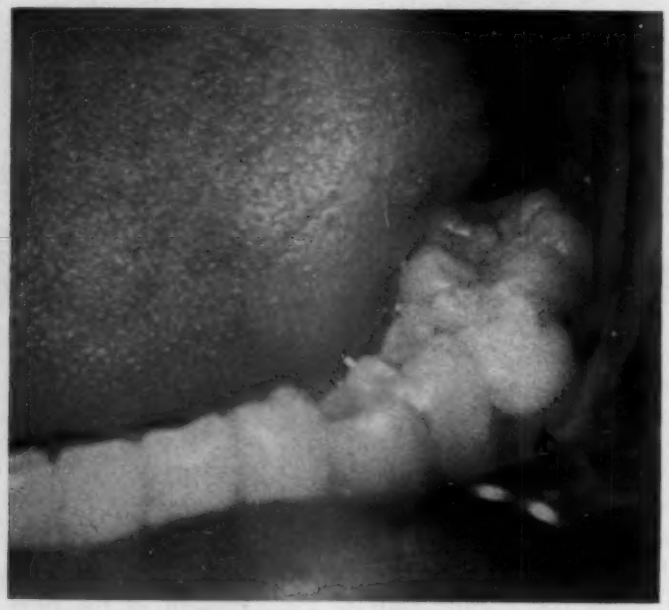


Fig. 5.—Mulberry (Pfluger's) molar in girl with congenital syphilis. The crowns are "pinched in" at the cuspal region, and a nodular, mulberry appearance results.

notched (Fig. 4). The constriction of the molar cusps with a resulting mulberry shape (Fig. 5) is called Pfluger's or mulberry molar. As in other enamel hypoplasias the deciduous teeth are rarely disturbed and only when the dystrophy occurs at a very early age. The maternal protection appears especially effective during formation of the deciduous teeth. While Hutchinson's teeth are characteristic of congenital syphilis, it is possible for other disturbances in amelogenesis similarly to affect the teeth, and such teeth cannot be taken, alone, as proof of congenital syphilis.

Another variant of hypoplastic teeth is the so-called Turner tooth (Fig. 6). In instances of periapical infection of the deciduous teeth, one, two, or three adjacent permanent tooth buds may be affected to produce unilateral hypoplasias, as opposed to the bilateral hypoplasias from systemic factors.



Fig. 6.—Turner teeth. The maxillary right first incisor and left first and second incisors are hypoplastic. The deciduous left first incisor had been injured by a fall during infancy and was the site of recurring "gum boils." Apparently, the periapical infection disturbed the three adjacent permanent tooth enamel organs. All other permanent teeth were clinically, or radiographically, hypoplasia free. A physician had prescribed vitamins and calcium to repair or prevent hypoplasia in the unerupted (normal) teeth.

b. *Hypocalcification.*—As has been pointed out, the enamel is formed in two stages. Disturbances from any cause during the second stage, hypocalcification, may result in cloudy or white spots on the teeth (Fig. 7). These spots are smooth and slick to the explorer. They must be differentiated from decalcified areas which are usually slightly softened and rough. However, decalcification sometimes occurs in cresent-shaped areas incisally to the cervical margin during eruption and then becomes arrested and even hardened when eruption carries them into self-cleansing areas. These arrested areas of decalcification will usually show roughening as opposed to the perfectly smooth areas of hypocalcification. In addition, hypocalcification almost always is bilaterally distributed, whereas decalcification may or may not be bilateral.

Chronic endemic dental fluorosis or mottled enamel is a hypocalcification resulting from excess fluoride ingestion during the period of amelogenesis. Mild dental fluorosis may be indistinguishable from hypocalcification caused by other agents. The history of residence during the first eight years of life is often the only evidence that will indict fluorine in such individuals. More severe mottling usually becomes stained brown, orange, or yellow (Fig. 8). Several methods have been proposed for removing the stain. The Ames method[6]

does not produce excessive decalcification. It may be followed by impregnation with celloidin, as suggested by Gore[7] for incipient caries, and the restaining will be greatly retarded or inhibited.

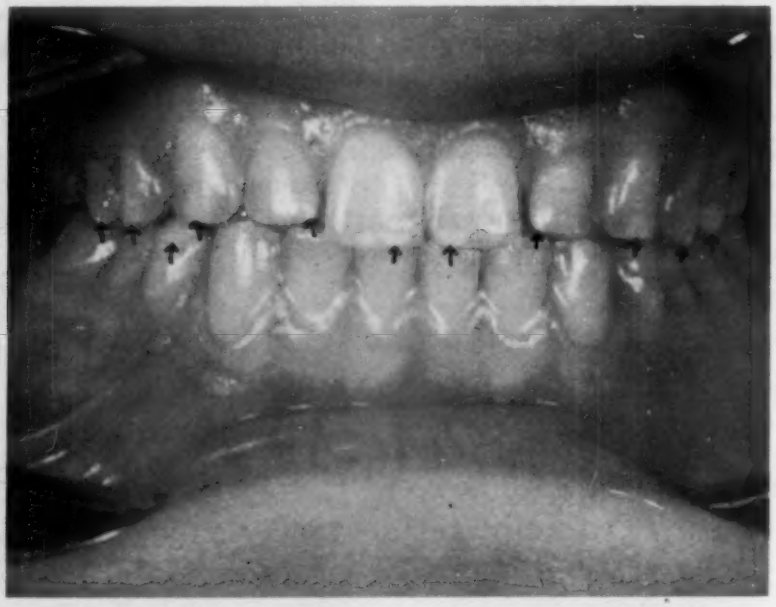


Fig. 7.—Hypocalcification. Clouded, white spots appear with smooth surfaces on the labial surfaces of the teeth.

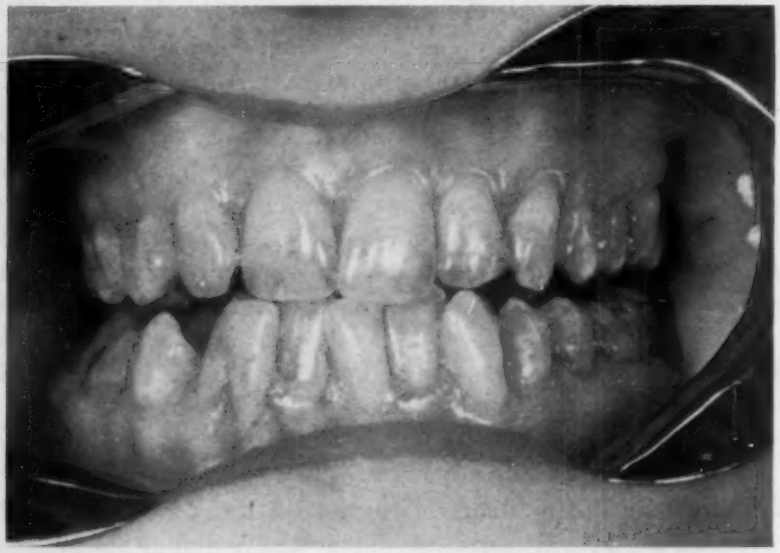


Fig. 8.—Chronic endemic dental fluorosis (mottled enamel). This young man shows the typical signs of severe mottling. The labial surfaces of the teeth show bands of paper white and various brown and yellow shades. Note also slight hypertrophy of traumatic origin (orthodontic bands) remaining in lower labial gingivae.

c. *Dentinogenesis Imperfecta.*—Dentinogenesis imperfecta (hereditary opalescent dentin) is an hereditary disease which results in imperfect formation

of dentin.[8] The teeth appear opalescent, iridescent, or violet color (Figs. 9 and 10). They wear down rapidly so that at 15 years of age they may be attrited to the gingival margin, show complete or nearly complete calcification of their pulp chambers and canals (Fig. 11), and a hereditary history may be secured. The enamel chips off readily, although it is not defective. The reason for this appears to be the relatively smooth dentinoenamel junction (Fig. 12) probably combined, as suggested by Gottlieb,[9] with a lack of odontoblastic processes to cross into the enamel.

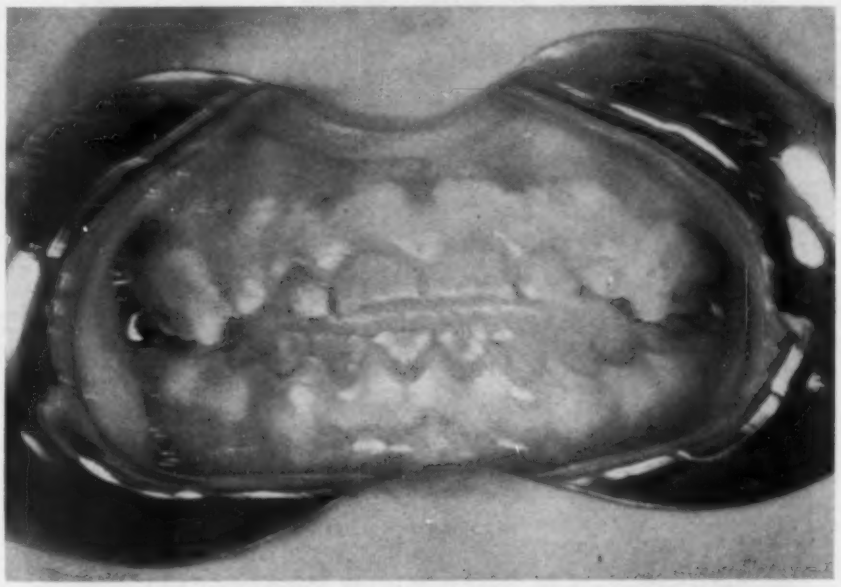


Fig. 9.—Dentinogenesis imperfecta in 12-year-old girl. The teeth are violet and opalescent and worn down markedly. A radiograph of her teeth is seen in Fig. 11. Her mother and one brother of three siblings showed the same inherited defect.



Fig. 10.—Dentinogenesis imperfecta in 6-year-old boy, brother of girl in Fig. 9. The deciduous teeth are practically worn to the gingival margins, but the two newly erupted permanent teeth have not yet worn down. They do show a peculiar gray color, opalescence, and filling in of the pulp cavities.

Histologically, absence or deficiency of tubules is seen in the dentin, and the rapidly formed tissue has an osteoid appearance (Fig. 12). The dentin is softer and has a lower inorganic content than normal.

The hereditary pattern is that of a dominant hereditary Mendelian factor. Fifty per cent of the offspring will show dentinogenesis imperfecta, by chance. There is no sex linkage. A relationship to osteogenesis imperfecta (brittle bones) has been demonstrated.[10]

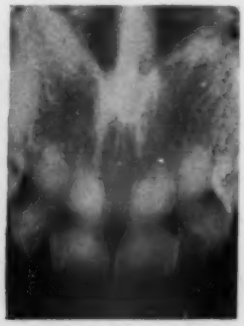


Fig. 11.—Radiograph of upper incisor region of patient with dentinogenesis imperfecta seen in Fig. 9. The pulp chambers and canals are obliterated and the roots are short. Some enamel is seen mesially and distally, differentiating this from enamel agenesis. (Radiograph from W. E. Koch, Jr.)

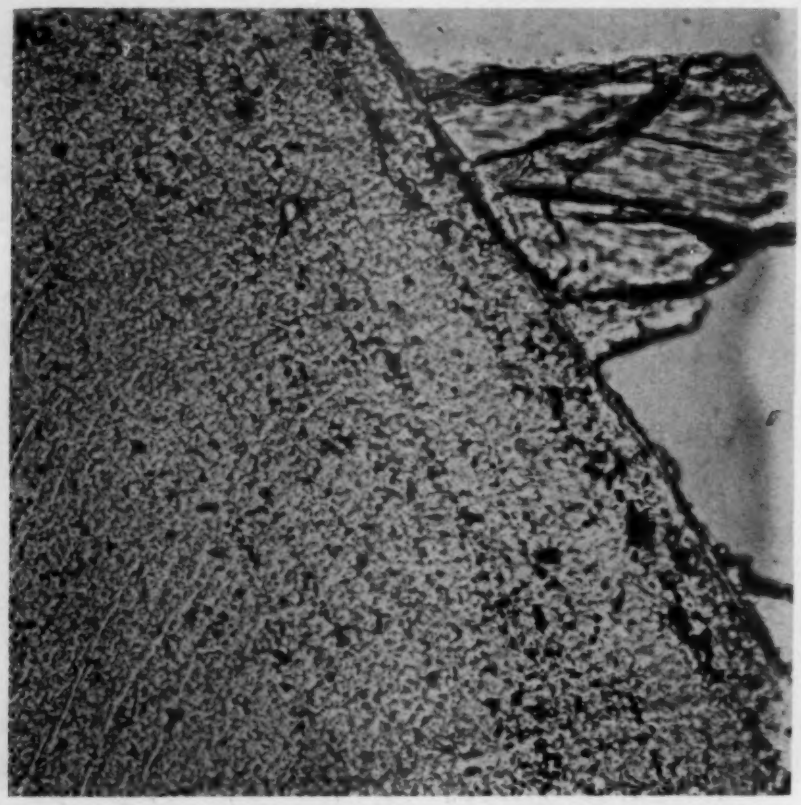


Fig. 12.—Section through dentinoenamel junction of tooth, showing atubular dentin of dentinogenesis imperfecta. Note large and numerous interglobular areas and smooth dentinoenamel junction. Remaining enamel is normal.

d. *Amelogenesis Imperfecta.*—Amelogenesis imperfecta bears the same relationship to the epidermally derived enamel that dentinogenesis imperfecta bears to the mesodermally derived dentin. The enamel is malformed. This disease, characterized by imperfectly formed enamel, is inherited as a Mendelian dominant, not sex-linked. As in dentinogenesis imperfecta both the deciduous and the permanent teeth are affected.

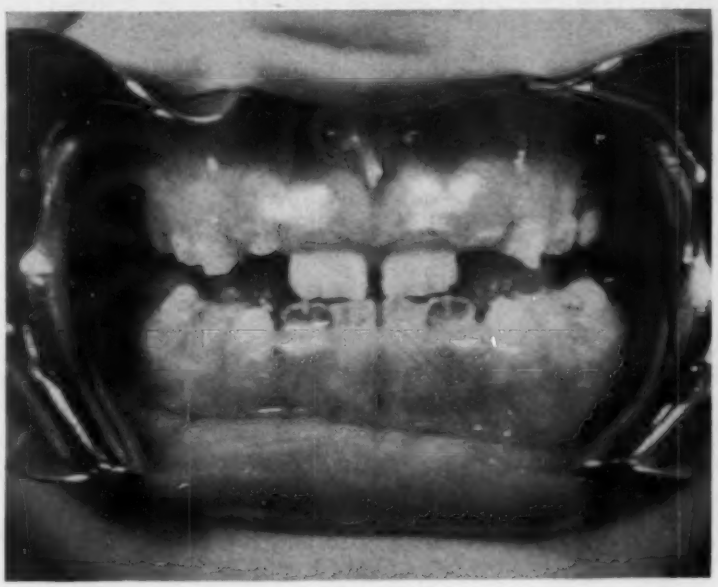


Fig. 13.—Seven-year-old girl with amelogenesis imperfecta of thin, flinty type.

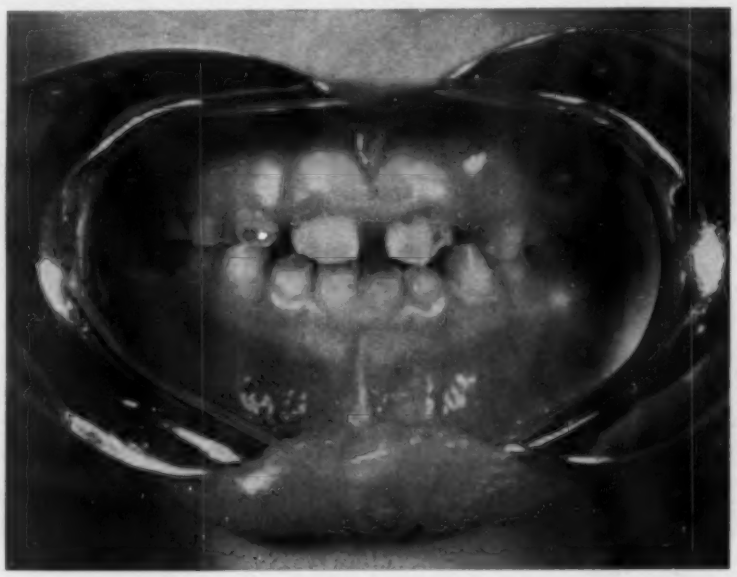


Fig. 14.—Six-year-old brother of patient seen in Fig. 13, showing identical type of amelogenesis imperfecta. The disease was not established in either parent in this family, but it was not possible to examine the father's mouth.

Macroscopically, two chief types are seen. In one the enamel is rather thick and stained a dark brown. The enamel appears to be softer than normal, clinically. In the second type the enamel is very thin, flintlike, and almost transparent (Figs. 13 and 14). It may be so thin as to break off at the incisal edges. In both types the pulp chambers and canals are normal.

e. *Agenesis of Enamel.*—This condition is relatively rare but dentinogenesis imperfecta is often misdiagnosed as enamel agenesis on casual examination. The disease is characterized by total lack of enamel formation. The underlying dentin is normal and the pulp chambers and canals are reduced only to a size corresponding to the amount of trauma induced by the exposure to oral fluids. Studies have shown a dominant Mendelian pattern of inheritance.

In dentinogenesis imperfecta, amelogenesis imperfecta, and agenesis of enamel the application of porcelain or acrylic jacket crowns in the anterior and full cast crowns in the posterior may preserve the teeth for years of service.

f. *Abrasion.*—Abrasion of the teeth is a slow and gradual wearing away of the teeth by friction other than that of mastication[11] (see *i. Attrition*). While occupational habits such as in glass blowing, sandblasting, needlework, carpentry, etc., may cause defects in the teeth, the most common cause of abrasion is the use of abrasive-containing dentifrices. In the days of Miller's study, dentifrices were abrasive enough to expose the pulp in eighteen hours of brushing, even cutting through enamel. The more modern dentifrices will not cut enamel but will readily cut cementum and dentin. Since Kitchin[12] has shown that 42 per cent of individuals have "critically" exposed root surfaces at the ages of 40 to 49 years, it is evident that many adults are exposing their teeth to danger of abrasion by dentifrices. A recent study[13] showed that the four leading sellers among powder dentifrices could cut halfway through an upper canine in 7.8 to 17 years of average[14] brushing, and that the four leading sellers among paste dentifrices would take from 28 to 117 years to cut halfway through the upper canine.

Since only the softer dentin and cementum are subject to the abrasion by dentifrices, there is a definite line of demarcation at the amelocementum junction (Figs. 15 and 16). This important fact, i.e., that the ordinary commercial dentifrices will not abrade enamel, serves to differentiate erosion in which the enamel is primarily removed. It appears that any type of brushing can remove the dentin, but cross-brushing is probably more effective.

Thoma has shown instances where the dental floss produced grooves in combination with the common paste dentifrice,[15] and, obviously, the toothpick can likewise carry abrading dentifrices to produce abrasion.

g. *Erosion.*—Erosion, as defined by Miller,[11] is a superficial chemical disintegration of enamel. Since many efforts have been made to find the cause of erosion and the disease is still of unknown etiology, the definition of removal of enamel by a physicochemical process is probably acceptable. Among the causes cited for erosion are acid formation by small local glands, acid formation by stimulation in the gingival sulcus due to occlusal trauma, acid formation from food debris in the gingival sulcus, changes in the oral secretions (to acid or alkali), enzymatic activity of leucocytes, and defects in the enamel. Many

authorities postulate a softening by chemical activity and removal by brushing. Recently, McClure and Ruzicka[16] produced an erosion-like lesion in the teeth of rats fed excess citrate ions. We are now investigating this ion in normals and in mouths with erosion. A psychosomatic factor has been cited by many individuals who note nervous strains and similiar factors.

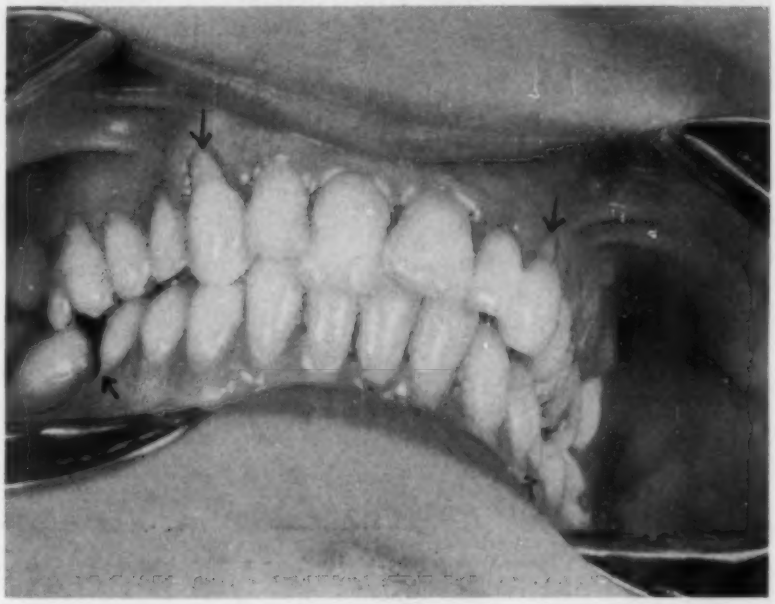


Fig. 15.—Abrasion on exposed roots of maxillary canines and other teeth. This patient used a commercial powder dentifrice vigorously for several years..



Fig. 16.—Marked abrasion on exposed roots of maxillary right canine and premolars. Note that the enamel has been undermined on those teeth. Another lesion is visible on the maxillary left second premolar. This patient used a commercial powder dentifrice for many years.

Clinically, smooth spots of enamel loss are seen. They may be wedge-shaped, cup-shaped, disk-shaped, irregular in form, L-shaped, U-shaped (Fig. 17) or they may simply show small round depressions or larger surface lesions

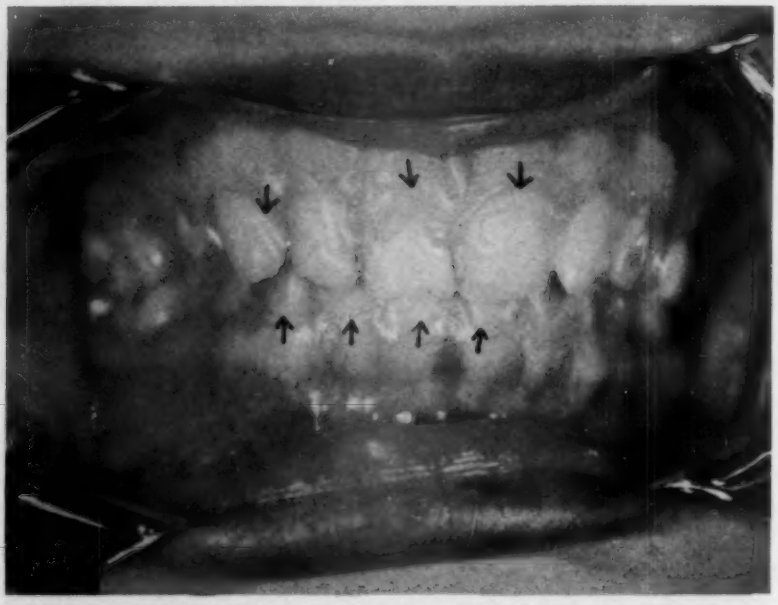


Fig. 17.—Erosion on labial and buccal surfaces of teeth in a young woman. This patient had marked occlusal trauma that could have been related to the erosion.



Fig. 18.—Erosion in the anterior teeth of a patient of early middle age.

(Fig. 18). The areas are hard and firm to the explorer and do not disturb the patient until the dentin is exposed, when they may produce marked hyper-

sensitivity to heat, cold, sweets, and acids. As noted previously, they begin, primarily, in the enamel.

h. *Acid Erosion.*—While acid conditions of the saliva have not been demonstrated as important factors in removal of tooth substance except in rare instances, regurgitated acid may produce loss of enamel, especially on the lingual surfaces.

The teeth show marked loss of enamel on the lingual surfaces (Fig. 19) and may be worn down to the gingival margin lingually and yet show good enamel structure labially and buccally.



Fig. 19.—Acid erosion of the lingual surfaces of the teeth of a patient who regurgitated her food for several years due to gastric disease.

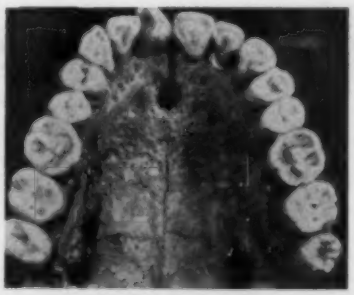


Fig. 20.—Marked attrition in the maxilla of a 25-year-old male Californian aboriginal. (From Leigh, R. W.: Dental Pathology of Aboriginal California, D. Cosmos. 71: 756, 1929.)

i. *Attrition.*—Attrition is the wearing away of tooth surfaces by friction of mastication. Obviously, there are three factors involved: the resistance of the teeth, the food abrasiveness, and time. In instances of poorly formed teeth, attrition may be marked in young individuals (Figs. 9 and 10). The use of very coarse foods in aborigines resulted in early attrition (Fig. 20). In our civilization we usually consider marked attrition a symptom of aging.[17] Ordinarily, attrition is considered as affecting only occlusal or incisal surfaces, but wear is also apparent on contact points with a resultant shortening of the length of the dental arch.

2. Lesions Which Enlarge the Gingiva

a. *Edema and Hyperemia.*—Edema and hyperemia may cause gingival pockets to become increased in depth without apical proliferation of the epithelial attachment. Such enlargement of the gingiva is due to accumulation of fluid in the tissue (edema) and increased vascularity (hyperemia). Either may occur alone or they may be observed concurrently.



Fig. 21.—Edema of maxillary gingivae and granulomatous masses (pyogenic granuloma) in mandibular gingivae of uncontrolled diabetic. Note drifting of teeth. Radiographs showed extreme alveolar atrophy.



Fig. 22.—Parulis in maxillary left mucobuccal fold of young man with periodontal abscess on premolar tooth. The face appeared swollen as in a cellulitis. The tissues have a purplish-red color.

The enlarged tissues in edema are soft and have a fluid appearance, as though one were looking into a pool of water. The overlying epithelium is usually thinned out and somewhat frangible (Fig. 21). In hyperemia the gingivae are red. The watery appearance of the edema may be obscured where both occur, as in early stages of Vincent's infection (ulceromembranous gingivitis).

b. *Inflammation.*—In gingival inflammation the tissues may be markedly enlarged by edema, hyperemia, and cellular infiltration. In addition, granulation tissue formation may result in so-called pyogenic granulomas (Fig. 21). Uncontrolled diabetes mellitus, for example, commonly leads to gingival and periodontal disease. The gingivae become deep purplish-red, enlarged, and somewhat dry. Proliferating pyogenic granulomas are characteristic, and loss of the alveolar bone results in wandering of the teeth. The flow of pus from the gingival sulci is marked. Burket[18] has pointed out that extensive alveolar destruction may precede other symptoms; he uses this as an argument in favor of routine urinalysis in all patients with periodontal disease.

Another form of gingival enlargement is caused by the parulis or "gumboil." It is usually found buccally or labially (Fig. 22) but may be found palatally from suppuration alongside a palatal root or on the palatal surface of a second incisor root. The mass is usually red, fluctuant, and tender. Careful exploration along the gingival sulcus will usually reveal an easily opened fistulous tract from which pus will flow freely. The parulis may be misdiagnosed for a tumor (Figs. 28 and 29), and vice versa. Gingival tumors are seldom fluctuant and do not evacuate pus.

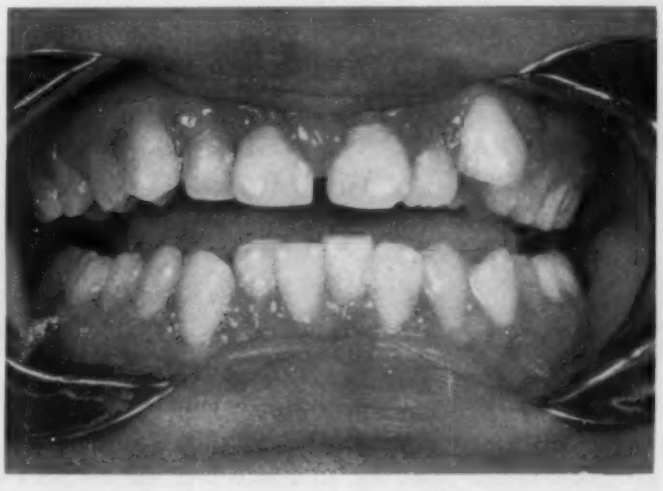


Fig. 23.—Hormonal hyperplasia in young woman during second trimester of pregnancy. The increased tissue is chiefly in interdental papillae subject to trauma. The tissues are reddish-pink and bleed readily.

c. *Hormonal Activity.*—During puberty, pregnancy, and sometimes the menstrual period, there may be an excess production of gonadotropic or estrogenic hormones. Ziskin, Blackberg, and Slanetz[19] observed the hypertrophic changes in these conditions and experimentally demonstrated their hormonal

background. Later, Ziskin[20] demonstrated a similiar hypertrophy in women receiving hormonal therapy. These conditions were earlier known as puberty gingivitis, gingivitis gravidarum, and gingivitis dysmenorrhea. A further proliferation in pregnancy produces so-called "pregnancy tumors."

Hormonal hypertrophy usually begins at interdental papillae subjected to trauma due to malposition of teeth (Fig. 23), or subject to inflammation from malhygiene. The tissues are usually fairly firm and highly vascular and hyperemic. Under pressure they will blanche, somewhat, and the color will return rapidly. The condition disappears at the completion of puberty. In pregnancy it usually arises between the third and sixth month and disappears after parturition. In the menstrual type it is recurrent. The pregnancy "tumors" are more localized forms of hypertrophy of the same type as the gingival hormonal hypertrophy.

Microscopically, the tissue shows marked increase in vascularity with some connective tissue proliferation. It resembles a hemangiofibroma.

In most instances the establishment and maintenance of good oral hygiene will control hormonal hypertrophy. Under irritation from calculus, rough or irregular restorations, sharp cavity edges, or food debris, inflammation, infection, and ulceration usually result. In the rare instances where the tissues do not recede at the end of puberty or at parturition, surgery is indicated, but it is usually contraindicated earlier.



Fig. 24.—Dilantin hyperplasia in young girl on sodium dilantin therapy for epilepsy. Redness of tissue was caused by a superimposed Vincent's infection. On control of the infection, the tissues assumed a normal pink color, were quite firm, and did not bleed easily.

d. *Drug Hyperlasia (Dilantin Hyperplasia).*—Dilantin sodium (diphenylhydantoin sodium) is used for the treatment of epilepsy. About 50 per cent of patients receiving this treatment show gingival hyperplasia within one year.[21] The hyperplasia may vary from very slight to massive type covering the entire crowns of the teeth. The true cause of the hyperplasia is unknown but poor oral hygiene accelerates the process.

The enlargement begins in the interdental papillae but soon bridges the tissue between the individual papillae (Fig. 24). The tissue is of fairly normal color, is firm, and the epithelium is somewhat thickened. It does not bleed readily, unlike hormonal hypertrophy. It usually is not sore or tender.

Microscopically, the tissue is composed of fairly dense fibrous connective tissue and may even show metastatic bone formation.

Conservative measures are indicated. Careful and proper toothbrush stimulation appears to control most cases if the patient will cooperate and if instituted before the connective tissue becomes too dense. If surgery is found necessary, it should be followed by early use of gingival stimulation. Reduction in the drug may aid but should not be requested unless absolutely essential. Prevention of the epileptic seizures is more important than control of the hyperplasia of the gingivae in practically all individuals.

e. *Trauma.*—Occlusal trauma may sometimes cause gingival hypertrophy or hyperplasia. Trauma from orthodontic bands may stimulate tissue proliferation (Fig. 8).



Fig. 25.—Acute leucemia in an elderly man. The gingivae are red, ulcerated, hypertrophied, and extremely sore. Establishment of maximum degree of oral hygiene with minimal operative trauma is imperative.

f. *Leucemias.*—The leucemias may result in gingival hyperplasia and ulceration of the oral mucosa. Oral pain is often the most aggravating subjective symptom. Whether the dyscrasias be myelogenous, monocytic, or lymphogenous, the same general oral symptoms occur. The gingival hyperplasia and ulceration may precede other symptoms.

Clinically, any ulcerative lesion which does not respond normally to therapy should be suspected as a possible leucemia. When such ulcerations are accompanied by hyperplasia of the gingiva and by general symptoms, such as malaise, fever, and pains in various parts, the suspicion should be strong (Fig. 25).

Obviously, leucemias may be mistaken for Vincent's infection. The oral necrosis in leucemia is identical with that of Vincent's infection, both being characterized by fusospirochetal and streptococcal invasion and an ulceromembranous (fibrinous) inflammation. The basis for the rapid destruction in



Fig. 26.—Patient with acute monocytic leucemia. Note enlarged submaxillary lymph nodes. This is due to leucemic infiltration. The skin was a yellow color, the lips were pale.

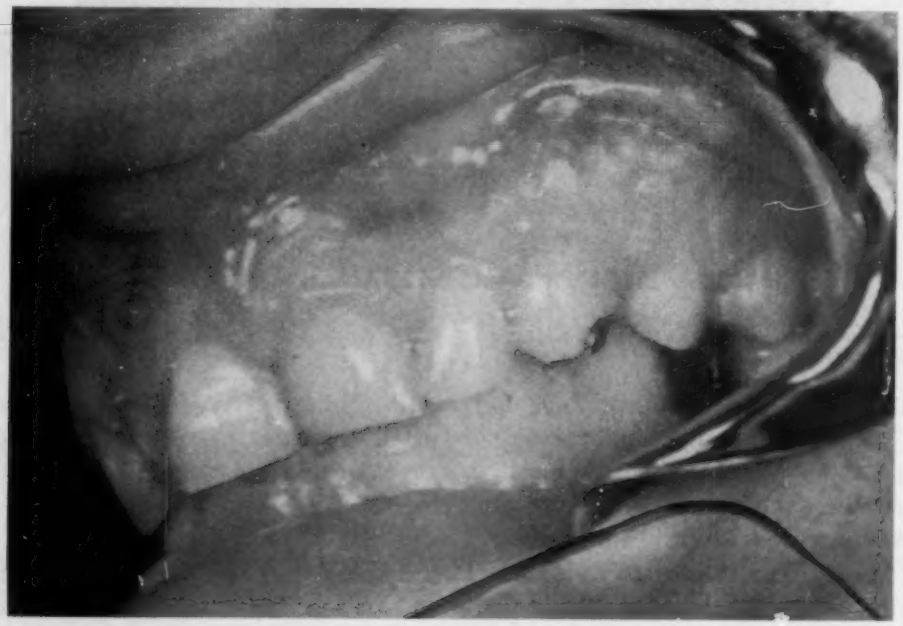


Fig. 27.—Intraoral view of patient seen in Fig. 26 after oral therapy. Before treatment the gingiva appeared worse than those of the patient seen in Fig. 25. The patient subsequently expired.

leucemia is almost total breakdown of the cellular defense mechanism. Careful hematological studies will be necessary to establish the diagnosis of leucemia. Since 87 per cent of monocytic leucemias, 40 per cent of myelogenous leucemias, and 23 per cent of lymphogenous leucemias present oral lesions,[18] the importance of this differentiation is seen. Granulocytopenia (agranulocytosis) may show similar oral lesions but a far different blood picture.

Treatment of oral lesions of leucemias should be principally establishment and maintenance of oral hygiene. This procedure will usually relieve the pain and control further necrosis. Irritating and caustic drugs are contraindicated. Hydrogen peroxide sprays (3 per cent) once per day and careful mouth washing after each meal are aided by local application of Mapharsen or penicillin. Gross calculus can be carefully removed after a few days of this therapy, and, during one of the patient's better periods, nontraumatizing prophylaxis may be completed (Figs. 26 and 27). Surgery is contraindicated, but in cases of absolute necessity it may be preceded and followed by transfusions.



Fig. 28.—Giant-cell epulis in 5-year-old boy. The tumor is of about one year's duration. It is purplish-red in color and shows ulceration where traumatized by the mandibular teeth.

g. *Tumors.*—Benign or malignant neoplasms may enlarge the gingiva. When a benign tumor occurs on the gingiva, it is known as an epulis, whether it be fibroma, papilloma, giant-cell tumor, or some other pathologic type. In 16 biopsies of gingival tumors in our laboratory, the following distribution was noted: Giant-cell tumor, 6; fibroma, 5; papilloma, 3; epithelioma, 1; sarcoma, 1. While, in general, fibromas are firm, pink, and covered with normal epithelium, papillomas firm but with a rougher paler surface, and giant-cell tumors softer and purple (Fig. 28), the differential diagnosis must be based on the biopsy. In the instance of smaller neoplasms the entire mass may be removed and used for microscopic examination, but, for larger tumors, a piece at least 0.5 by 0.5 by 0.5 cm. should be cut from the growing edge and the surrounding



Fig. 29.—Metastatic lesion of osteogenic chondromyxosarcoma originating in leg of 12-year-old boy. The mandibular lesion was first diagnosed as parulis (Fig. 22). The similarity to epulis (Fig. 28) is also marked. The lesion was excised, locally, four times. Metastasis spread from the leg to the shoulder and other parts before death.



Fig. 30.—Radiograph of mandibular second molar in patient seen in Fig. 29. The total loss of alveolar support is evident. The tooth was quite loose, a symptom which should suggest the possibility of tumor growth.

normal tissue. The material should not be roughly handled and should be dropped immediately into 10 per cent formalin or other suitable fixative. The material should be sent with complete history to a competent oral pathologist for examination.

Malignant tumors may occur on the gingivae and simulate benign tumors or inflammatory reactions. A metastatic osteogenic chondromyxosarcoma of the mandible (Fig. 29) was originally diagnosed as a "gumboil" or parulis (see Fig. 22). Radiographs of teeth in the region of malignant tumors usually show marked loss of supporting bone (Figs. 30, 34, and 35).

h. *Scurvy.*—In scurvy the gingivae are adversely affected. They become hypertrophied, spongy, and bluish-red. They bleed readily and usually are sore. The teeth become loose and the condition may simulate a severe Vincent's infection. Bacteriologic study will show a profusion of spirochetes, fusiforms, and streptococci.

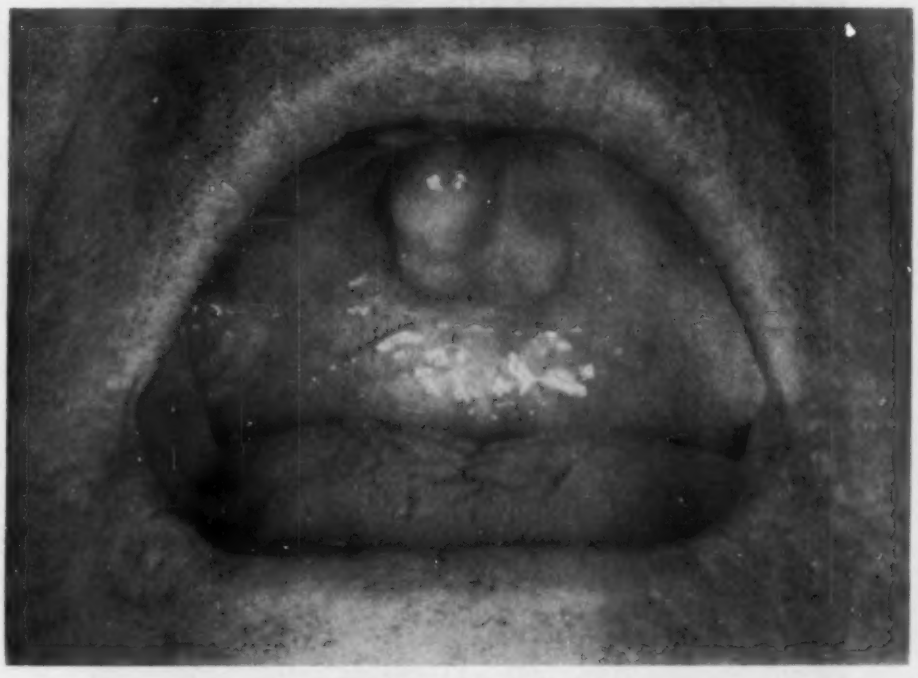


Fig. 31.—Torus palatinus of lobulated type.

3. NEOPLASMS AND OTHER SWELLINGS OF THE PALATE

a. *Torus Palatinus.*—Torus palatinus is a benign hyperostosis commonly observed in the midline of the posterior portion of the hard palate (Fig. 31). It is bone hard, covered by normal epithelium, and may be nodular, pedunculated, loculated, scaphoid, or spheroid. Tori vary greatly in size. Miller and Roth[22] observed them in 25 per cent of 1,000 patients of various ages.

They are painless and insignificant unless the overlying epithelium becomes traumatized or infected, or unless they interfere with denture construction. An occasional large torus may interfere with speech or mastication. Micro-

scopically, they show more or less dense bone growth. In the larger tori there may be spongiosa as well as cortical bone.

b. *Mixed Tumor of the Palate.*—Mixed tumors may occur in any location in which salivary glands occur. This includes not only the sublingual, submaxillary, and parotid region, but also the palate, lips, and alveolar ridge. They may invade the bone in the palate, and radical operations are often necessary.



Fig. 32.—Mixed tumor of the soft palate in 73-year-old man. (From Burford, W. N., Ackerman, L. V., and Robinson, H. B. G.: Am. J. Orthodontics and Oral Surg. [Oral Surg. Sect.] 30: 353, 1944).

They may appear, grossly, as small or large swellings on the posterior of the hard palate or on the soft palate (Fig. 32). They may be misdiagnosed as tori (Fig. 31).

Microscopically, they show epithelial acini, arranged in glandlike form, and surrounded by connective tissue with mucoid areas.

c. *Adenocarcinoma.*—Malignant tumors, unfortunately, are not rare in the mouth. Approximately 5 per cent of all fatal cancers originate in the tongue, lips, palate, cheeks, or jaws. The dentist must be alert to diagnose malignant tumors and to refer them for adequate treatment.

Malignant tumors may simulate benign growths and remain unnoticed until the chances of adequate treatment are greatly reduced. Early diagnosis is the most important single factor for successful tumor therapy. The adenocarcinoma illustrated in Figs. 33, 34, and 35 will demonstrate the need for thorough examination. The patient reported with a swelling of the palate (Fig. 33). She had been treated for periodontal disease for several months. It was immediately sus-

pected that the tooth had pulp disease, and periapical infection was producing a parulis (mesial root, Fig. 34). Radiographic study showed loss of normal architecture and a "snowflake" appearance. The spiked appearance of the roots is characteristic of teeth in regions of malignant neoplasms (Fig. 35).



Fig. 33.—Adenocarcinoma of palate and maxilla. The epithelial covering was normal in appearance and the growth was very hard.



Fig. 34.—Radiograph of maxillary first molar region of patient seen in Fig. 33. Note incidental periapical rarefaction on the mesial root and loss of architecture of bone posterior to first molar. (Radiograph from W. E. Koch, Jr.)



Fig. 35.—Radiograph of second and third molar region of patient seen in Fig. 33. Note spiking of roots, loss of bone pattern, and "snowflake" appearance. (Radiograph from W. E. Koch, Jr.)



Fig. 36.—Hyperplasia and hypertrophy of palatal mucous glands resembling adenocarcinoma seen in Fig. 33. This lesion was softer and the radiographs revealed no loss of bone pattern.

While diagnosis of adenocarcinoma was made from microscopic examination of the surgical specimen, a preliminary diagnosis of probable malignancy was possible on the basis of the radiographic evidence.

d. *Hyperplasia of Palatine Mucous Glands.*—Benign hyperplasia may ape tumor growth. These usually give a history of extremely slow growth although the patient may have only discovered the swelling within a relatively short time.

Biopsy is important in establishing the differential diagnosis of these benign lesions. An example is shown in Fig. 36. This benign hyperplasia and hypertrophy of the palatine mucous glands was confirmed by biopsy. The radiograph showed no abnormality of the bone surrounding the adjacent teeth, but otherwise the clinical appearance was similar to that of the adenocarcinoma (Figs. 33, 34, and 35).

4. HYPERPLASIA AND INFECTIOUS GRANULOMAS

a. *Hyperplasia and Hypertrophy.*—Hyperplasia and hypertrophy of the edentulous ridge may occur under ill-fitting dentures, as a result of trauma or along the margins of irregular extraction scars. In many instances these hyperplasias and hypertrophies will be reduced without surgical intervention if the irritation is removed. If recession fails to take place, surgery may be necessary.



Fig. 37.—Hyperplasia of mucosa along line of extraction scar. This resembles the mouth from which the actinomycotic colony in Fig. 38 was taken.

An example of a hyperplasia along the irregular margins of an extraction wound is shown in Fig. 37. While this is obviously only an excess tissue growth, the removed material should be examined by a competent oral pathologist. The following case will serve as an example of the need for such examination.

b. *Actinomycosis.*—A woman about 45 years of age had her lower anterior teeth removed before construction of a full denture. The teeth were involved by periodontal disease. Three weeks following extraction she reported with an appearance almost identical with that in Fig. 37. It was considered simple hyperplasia and was not photographed. The tissue was surgically trimmed and the removed material sent for microscopic examination. Under the microscope, colonies of actinomyces were observed (Fig. 38). There were no symptoms of actinomycosis such as multiple fistulas, "sulfur granule" pus, etc., but the patient was referred to the department of medicine where a prophylactic course of penicillin and sulfonamides was instituted. The routine pathologic examination may have resulted in abortion of a possible severe actinomycosis of the jaw.

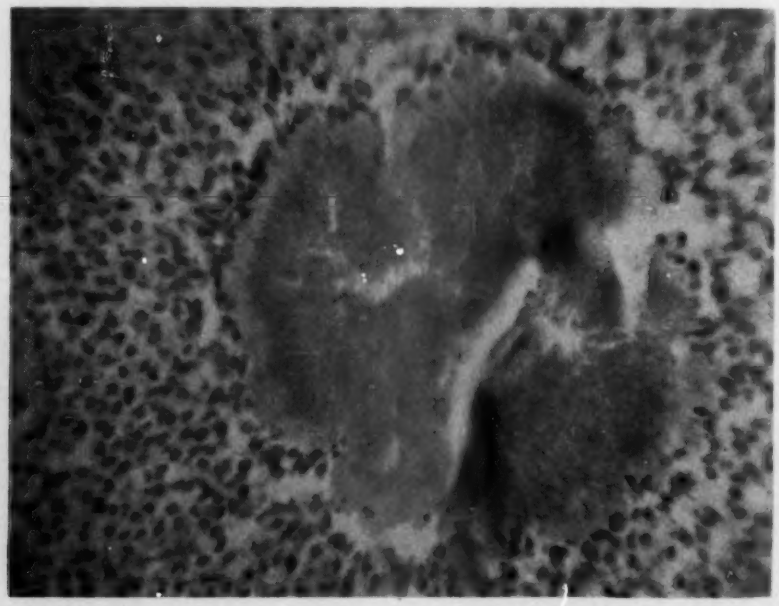


Fig. 38.—Actinomycosis colony from hyperplastic tissue along healing extraction scar.

5. LESIONS OF THE BUCCAL MUCOSA

a. *Leucoplakia.*—Leucoplakia is characterized by hyperkeratosis of the oral epithelium. It is the equivalent in the mouth of a corn on the toe. It is caused by mechanical, thermal, chemical, or electrical irritation (Figs. 39 and 40) on a predisposing background of individual susceptibility. Hormones, vitamins, and infections such as syphilis have been indicated as causes by some authorities, but, for the most part, the evidence has been inconclusive. Tobacco smoking in a susceptible individual is probably the greatest single cause of leucoplakia.

Clinically, the oral mucosa loses its normal translucency, becomes thickened and roughened. It has a leathery appearance and feel, has irregular margins, and is almost invariably nontender and painless. It may occur on any area of the oral mucosa in addition to the cheek. About 50 per cent of the leucoplakia is located on the buccal mucosa, and the rest is on the tongue, palate, lips, gingiva, and floor of the mouth. Burket[18] has suggested the use of Lugol's

iodine to differentiate leucoplakia from other lesions. Leucoplakia does not stain mahogany brown and stands out from the surrounding mucosa.

Microscopically, a hyperkeratinization is evidenced in leucoplakia.



Fig. 39.—Leucoplakia of buccal mucosa in elderly patient. The lesion is white, opaque, and rough. (From Ellis Fischel State Cancer Hospital, Columbia, Missouri.)



Fig. 40.—Leucoplakia in floor of mouth of patient with relatively rare electrogalvanism. The opaque white appearance is evident.

Removal of the irritant is usually the most effective treatment. Although leucoplakia has often been called a "precancerous" lesion, it seldom progresses to true carcinoma. However, on occasion, persistence of the irritant may lead to anaplasia and tumor growth. The patient shown in Figs. 40 and 41 presents two rare circumstances. His leucoplakia was caused by irritation from electrogalvanic current flowing between dissimilar metals as well as smoking, and he developed a fatal carcinoma. He had a large gold crown on the lower left

first molar and amalgam fillings in the lingual surfaces of his lower canines, premolars, and incisors. Current, as measured by a galvanometer, was flowing between the gold and silver amalgam at a rate 16 times as great as in other mouths with dissimilar metals tested at random. The gold crown showed a fine white metallic film and the amalgam fillings were pitted. Electrogalvanism, such as this, is rare. It was suggested that the patient have the fillings replaced by nonmetallic plastics and stop smoking. After some procrastination the teeth were removed but smoking was not eliminated. Four years later the patient was operated by Dr. V. P. Blair for advanced carcinoma of the region (Fig. 41), but he had waited too long to expect success from surgery.



Fig. 41.—Squamous-cell carcinoma (*A*) developed in floor of mouth of patient seen in Fig. 40. This is a relatively rare occurrence. Note leucoplakia is still present (*B*).

b. *Lichen Planus.*—Lichen planus is a dermatological disturbance which shows oral lesions in about 50 per cent of the cases. The disease is more common in females than in males. The skin lesions are violet-colored groups of glistening papules and are found principally on the flexor surfaces of the wrists and forearms and on the ankles. The oral lesions occur either without dermal lesions or preceding or accompanying dermal lesions.

The oral lesions occur as fine reticulated white lines radiating in lacelike pattern from small white dots. The lines and dots have a pearly appearance and are found chiefly on the buccal or labial mucosa (Fig. 42). The lesions rarely ulcerate, unlike herpetic lesions, and are smooth and slick, unlike leucoplakia. Lingual lesions may resemble benign migratory glossitis. Lichen planus, intraorally, is painless, but patients sometimes complain of itching in the mucosa. Gastric upsets or nervous disturbances may precede the lesions, which may disappear spontaneously to recur at the occasion of another gastric or nervous disbalance.



Fig. 42.—Lichen planus on buccal mucosa of young man. The lesion is smooth, pearly bluish-white, reticulated, and painless. Lichen planus was not observed extraorally. The oral lesions increased with gastric distress at times of nervous tension.

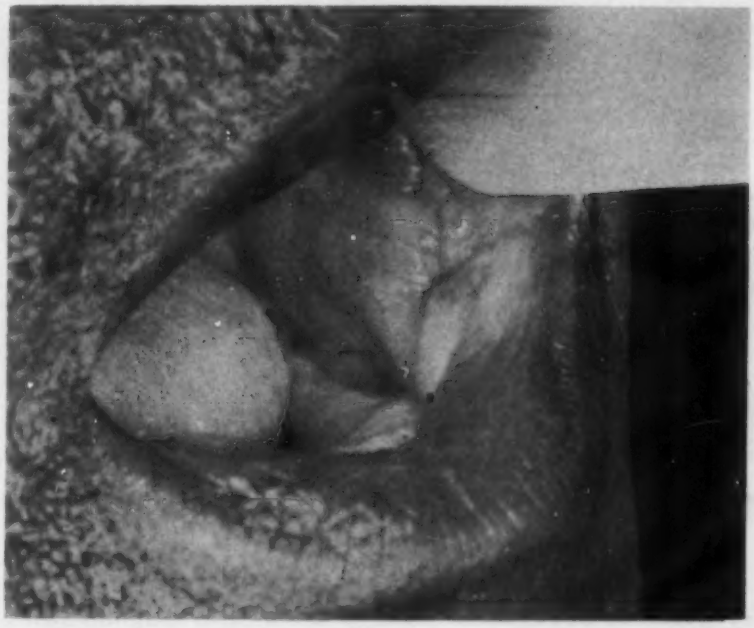


Fig. 43.—Negroid parakeratosis of buccal mucosa. The mucosa is thickened, smooth, and bluish. This is probably a normal racial characteristic.

Microscopically, the epithelium is somewhat thickened and edematous. Collections of lymphocytes are pronounced just beneath the basement membrane.

Treatment consists of keeping the oral cavity clean and protecting the lesions from trauma.

c. *Negroid Parakeratinization of the Buccal Mucosa.*—In the Negro, a pearly gray parakeratinization of the buccal mucosa is observed so frequently that we are led to assume that this is normal for the race. The buccal mucosa is pearly, gray, slightly thickened, and smooth (Fig. 43). It may be mistaken for lichen planus.

d. *Cheek Biting.*—Cheek biting is often observed in nervous individuals. The line of trauma may be at any place on the cheek opposite the occlusal line. Grayish-white areas of eroded epithelium are seen, with ragged surfaces from which epithelial cells can be teased or plucked with a tissue forceps (Fig. 44). There may be pain as a result of breaking through the epithelium. Secondary infection by oral organisms may lead to a typical ulcer.



Fig. 44.—Irregular, traumatized lesion of cheek biting. The surface is ragged and the borders irregular. It is white, tender, and the epithelium can be peeled or teased off readily.

6. PIGMENTATION OF THE GINGIVA

a. *Heavy Metal Intoxication.*—Intoxication with bismuth salts, lead salts, or mercurial salts may lead to pigmentation of the gingival margin. The bismuth line is seen principally in individuals receiving bismuth therapy for syphilis, the lead line chiefly in workers in trades utilizing lead (lead smelting,

painting, brass foundries, etc.), and mercury pigmentation in individuals receiving the drug or contacting the metallic salts in their trade.

The typical bismuth line is found at the gingival margins (Fig. 45). It is more pronounced in areas of inflammation, probably due to the attraction of the blood-borne particulate matter to the areas of inflammation (anachoresis) as well as to the presence of sulfides which convert other bismuth salts to bismuth sulfide. In mouths with gross malhygiene and under heavy dosage, frank bismuth stomatitis may occur. The lead line is not as well demarcated as is the bismuth line but is quite similar in appearance. The pigmentation of mercury is less common and, since the mercury is more toxic to the tissues, severe stomatitis often results.

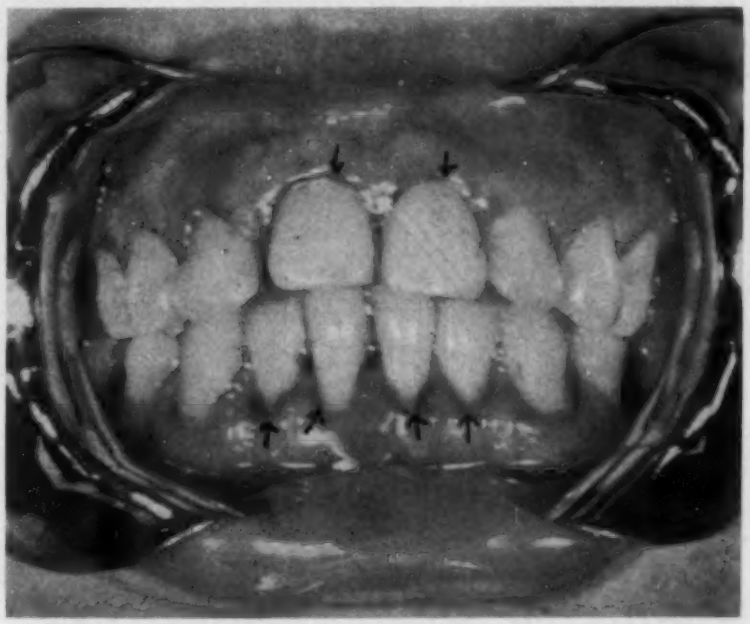


Fig. 45.—Mild bismuth line on anterior teeth. The paper corner test is recommended to differentiate from pigmented calculus.

The use of 30 per cent hydrogen peroxide has been suggested for decolorizing the bismuth line.[23]

b. *Calcareous Deposits.*—Subgingival calculus with pigmentation from tobacco, drugs, blood pigments, or foods may show through the gingiva and appear as gingival pigmentation (Fig. 46). A simple test, which we first heard described by Dr. Thomas Cook, consists of passing the corner of a piece of white paper into the gingival sulcus. If the deposit is on the tooth it will be blotted out and the gingivae will appear normal, but if the deposit is in the gingivae it will continue to show over the paper.

c. *Negro Pigmentation of the Gingiva.*—The Negro commonly shows pigmentation of the gingiva which may be mistaken for metallic lines. Dummett[24] has described the distribution of these pigmentations and their occurrence. Figs. 1 and 5 show negroid pigmentation of gingiva.



Fig. 46.—Pigmented calculus on anterior teeth. If subgingival calculus were left after partial prophylaxis, it would show through and resemble bismuth or lead line.



Fig. 47.—Ankyloglossia. Note that the frenum is attached near the tip of the tongue.

7. ANKYLOGLOSSIA AND HYPERMOBILITY OF THE TONGUE

a. *Ankyloglossia.*—Tongue-tie is due to anterior attachment of the lingual frenum rather than to a short frenum, as is commonly described. The frenum binds the tip of the tongue and interferes with normal speech (Fig. 47). Its surgical correction is indicated.

b. *Hypermobility of the Tongue.*—Hypermobility of the tongue results from posterior attachment of the lingual frenum. It may be of serious consequence in infants because of possible tongue swallowing and subsequent suffocation. A

large tongue that is hypermobile is illustrated in Fig. 48. The patient is able to "swallow" the tongue voluntarily (Fig. 49). This "swallowing" appears to be placing of the tongue behind the soft palate and into the nasopharynx. It is apparent that such an accident in an infant might lead to death.

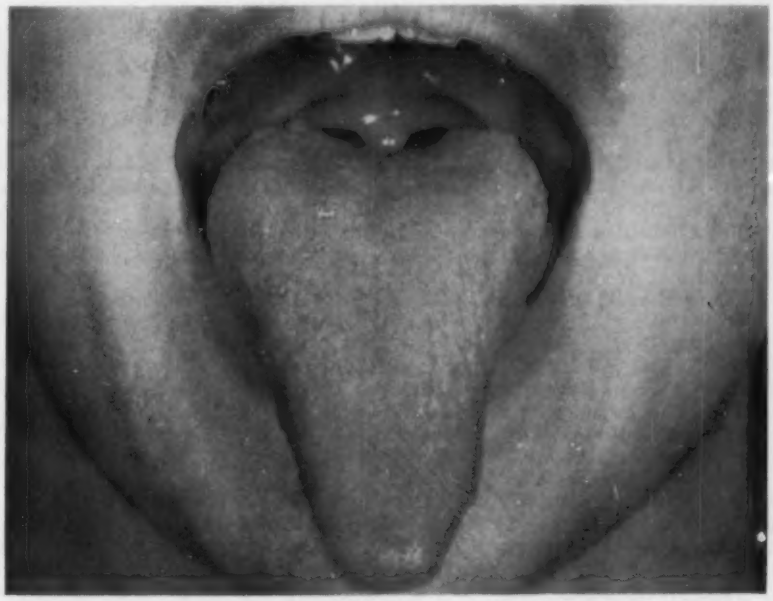


Fig. 48.—Extremely long and hypermobile tongue.



Fig. 49.—Same tongue as that seen in Fig. 48 "swallowed" by placing behind soft palate in nasopharynx.

SUMMARY

A series of cases of oral-medical and oral-surgical interests are presented to demonstrate the importance of history taking, careful clinical examination, radiography, and biopsy in differential diagnosis. Lesions of the hard tissues, gingivae, palate, buccal mucosa, and tongue are used as examples.

The author wishes to express his appreciation to his former colleagues at Washington University (St. Louis), especially Drs. Lester Jasper, W. E. Koch, Jr., Joseph Laffler, Ruth Martin, and James Rose, and his present colleagues at Ohio State University, especially Drs. S. S. Arnim, Virgil Carnes, P. C. Kitchin, Phillip Mahan, D. P. Snyder, Harry Spangenberg, and Claude S. Wright, and Miss Dorothy Permar, without whose assistance it would have been impossible to collect this material.

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ODONTOGENIC TUMORS

A Survey of Seventy-Five Cases

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INTRODUCTION

ODONTOGENIC tumors, derived from dental or potential dental tissue, are common and have been discussed extensively in the literature. However, there has been no uniformity in classification of these tumors, nor has it been suggested that these tumors may arise in conjunction with one another because of the inductive influence of one tissue on another in their pathogenesis. The purpose of this report is threefold: (1) to formulate a classification of odontogenic tumors; (2) to describe their structure; and (3) to illustrate the inductive effects of one tissue on another in the production of odontogenic mixed tumors. The material used in this study comprises 75 cases of odontogenic tumors, some of which had been previously studied by one of us (H. M. G.) at the Registry of Dental and Oral Pathology, Army Institute of Pathology.

CLASSIFICATION

We are in accord with the classification of odontogenic tumors based on structure; however, certain terms are used in this paper with modifications in meaning. "Odontogenic fibroma" is used to indicate the soft mesenchymal tumor, while "dentinoma" is reserved for the pure mesenchymal tumor composed of islands of dentine in a connective tissue stroma. "Odontoma" is regarded as a mixed tumor made up of epithelial and mesenchymal elements and is spoken of as "odontogenic mixed tumor." Three types are recognized.

The following classification is the one adopted by us:

I. Epithelial tumors (36 cases studied)
   1. Adamantoblastoma
   2. Enameloma

II. Mesenchymal tumors (15 cases studied)
   1. Odontogenic fibroma
   2. Dentinoma
   3. Cementoma

III. Odontogenic mixed tumors (odontomas) (24 cases studied)
   1. Soft odontoma—epithelium and mesoderm
   2. Soft and calcified odontoma—adamantoblastoma arising in conjunction with a forming or completely formed odontoma; all sorts of histologic variations due to the inductive effects of one tissue on another
   3. Completely formed odontoma with enamel, dentine, pulp, cementum, periodontal membrane
      a. Compound (many small teeth)
      b. Complex (irregular tooth structure)

From the Dental Department, Massachusetts General Hospital, Boston, Mass.

Tumors may arise from either odontogenic epithelium or mesenchyma and may be either soft or calcified. The soft epithelial tumor is commonly called adamantoblastoma; the calcified, enameloma. The mesenchymal odontogenic tumor arises as a fibroma; in the calcified stage, if dentine is produced, it is called dentinoma; if cementum, cementoma. Tumors may also originate from both the odontogenic epithelium and mesenchyma. These are known as odontomas or odontogenic mixed tumors and may belong to any one of three groups: (1) soft ondontomas, (2) soft and calcified odontomas, and (3) completely calcified odontomas.

EPITHELIAL TUMORS

Adamantoblastoma.—The adamantoblastoma is an epithelial odontogenic tumor in which the cells may differentiate to a variety of forms depending to some extent on the development of the epithelium at the time when tumor formation begins. Solid and cystic types are distinguished, both being of a slowly growing, benign nature, to judge from clinical observation. The frequency of recurrence after operation is due rather to the difficulty of removing the entire lesion by conservative operation than to any malignancy of the tumor; however, several cases recorded[1, 2] indicate that it can be malignant.

General characteristics: Microscopically, the tumor may appear solid, cystic, or a combination of both. If solid, it is composed of cords or strands of epithelium growing in a connective tissue stroma, or occasionally almost without stroma. The epithelial cords, which may form a network or take on a papillary structure, may resemble the anlage given off from the dental lamina and have a similar tendency to form small buds comparable with the earliest stage of the enamel organ. In the central portion of the cord or lobule the epithelium appears stellate, like the stellate reticulum of the enamel organ; in the periphery the cells are cuboidal to cylindrical.

The cystic type is characterized by lobules or strands of adamantine epithelium with a peripheral layer of cylindrical cells which, in structure, approach ameloblasts. These cells lie on a basement membrane. The central portion consists of stellate to squamous epithelium undergoing cystic degeneration. Pressure of cystic fluid causes these spaces.

The adenoid adamantoblastoma is the least common and is composed of small epithelial cells arranged in acini which usually contain a mucoid substance. The oral epithelium has potentialities of forming glandular as well as dental structures and, since the components of the enamel organ are derived from the epithelium of the oral cavity, cells may be present which have the ability to differentiate into adenomatous structures. Tissue of this kind composed one of the adamantoblastomas studied and was present in three of the odontogenic mixed tumors. The acinar arrangement was characteristic and the mucoid material filling the acini seemed to attract calcium salts. The globular pattern of the calcification suggested calcospherites.

Variations in morphology: The well-differentiated adamantine epithelium is very frequently encountered. In a lobule of epithelium the peripheral cells

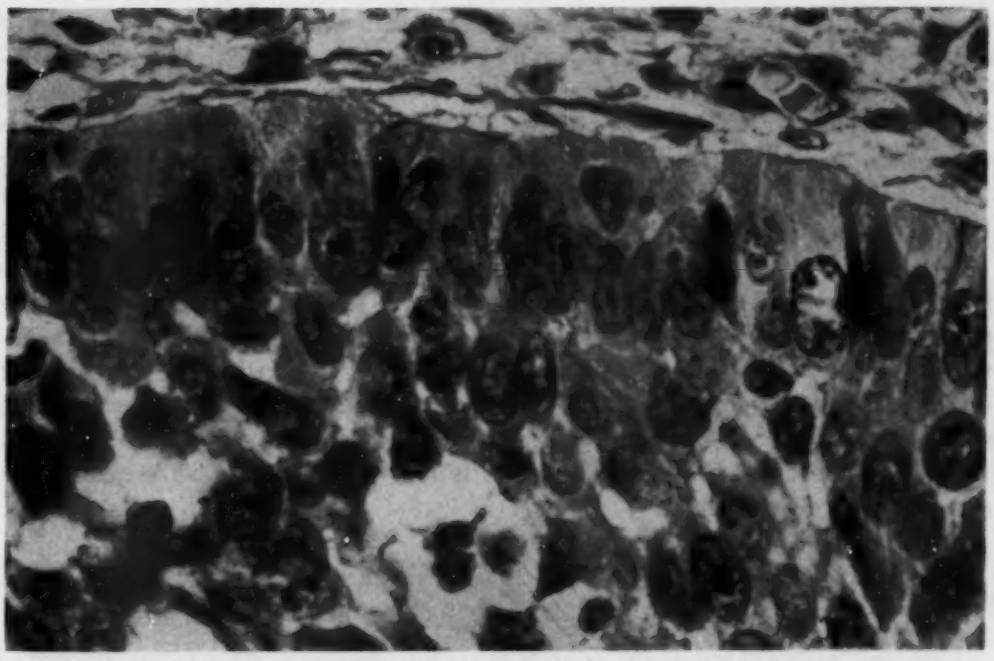


Fig. 1.—Peripheral cells of a strand of epithelium in an adamantoblastoma. The palisading of the columnar cells is characteristic of this type.

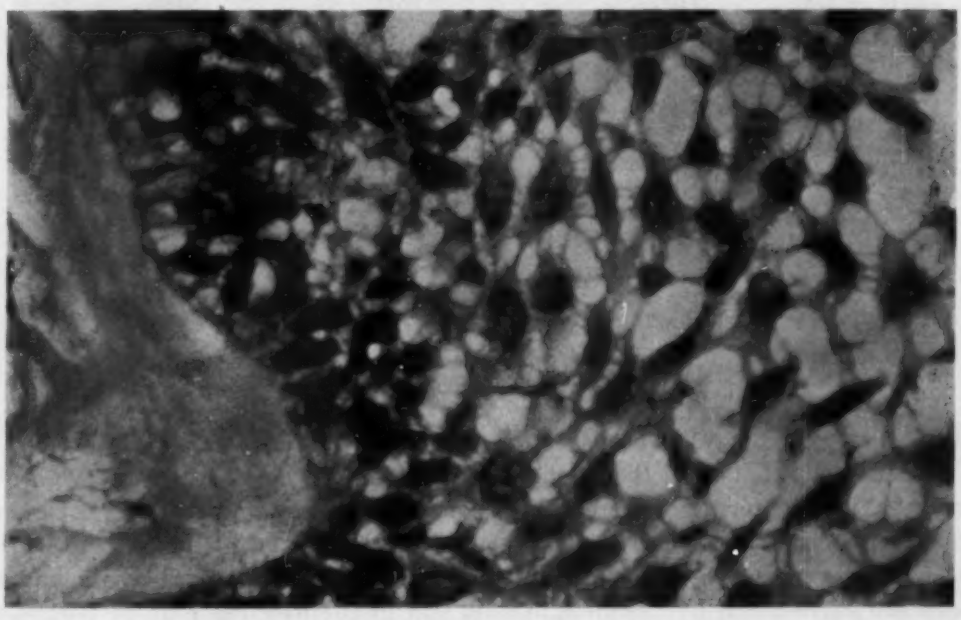


Fig. 2.—The central cells of an epithelial strand in an adamantoblastoma. Note the long slender anastomosing protoplasmic processes of the cells and also the spindle-shaped cells resembling fibroblasts.

are columnar in shape and are arranged in a palisade corresponding to the ameloblasts in the enamel organ of a three-month-old fetus (Fig. 1). Rarely, a distinct basement membrane is found between the epithelial strands and the fibrous stroma. The central portion of the epithelial lobule may consist of very loosely arranged stellate cells corresponding to the stellate reticulum of the enamel organ (Fig. 2), or a more squamous type of epithelium. The main feature, however, is the peripheral columnar cell with, for the most part, centrally placed nuclei.

A less differentiated adamantine epithelium is more common in the large group of cases we studied. The peripheral cells are cuboidal or even oval in shape with centrally situated nuclei; these cells have but little cytoplasm and may pile up in many layers, forming a vague multilayered zone. The central cells are usually more compactly grouped and tend to be spindle or oval in shape, sometimes being almost of the same character as the peripheral cells except for the arrangement. The peripheral cells lie perpendicular, while the central cells show no orientation.

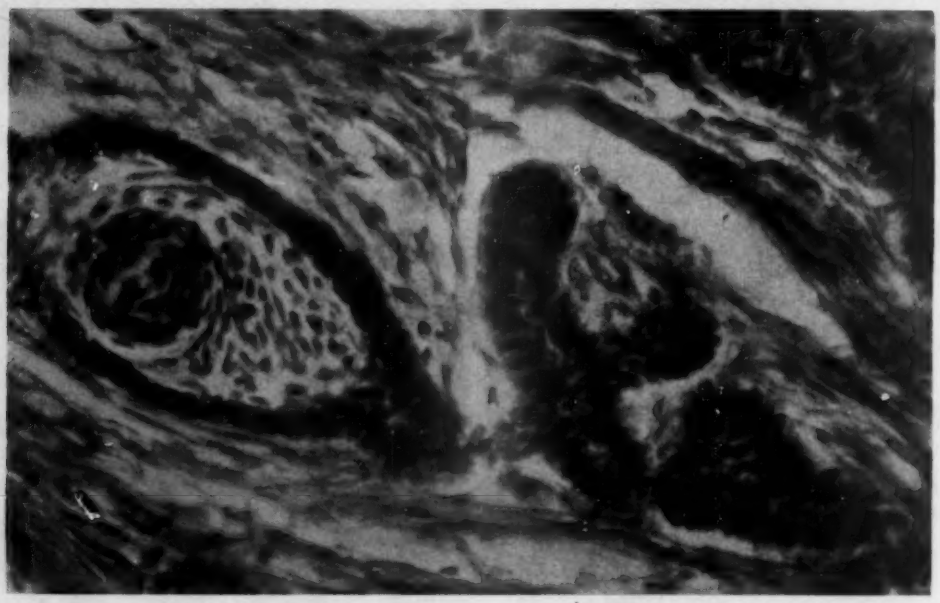


Fig. 3.—Squamous change in the central area of an epithelial lobule in an adamantoblastoma.

Squamous change of the epithelium is a frequent observation always beginning in the center of an epithelial strand spreading toward the periphery. The earliest change consists of a few squamous cells found in the central portion of the mass (Fig. 3); at times multiple foci may be noted. These squamous cells are surrounded by the stellate cells and in turn by the zone of peripheral cells. Frequently a strand of epithelium may consist wholly of squamous cells bordered by the peripheral cells.

The adenomatous arrangement of the epithelium in adamantoblastoma is rare, but since embryologically the oral epithelium has the potentialities of forming glands, one should expect a certain percentage of tumors examined to

have this type of epithelium. The cells are small, rather undifferentiated, dark-staining, with round or oval nuclei and with but a small amount of cytoplasm. They are arranged in acini or a follicular fashion, and homogeneous, pink-staining, and rather refractile mucoid material fill the spaces. This material could also be found in between and in the cytoplasm of the epithelial cells.

Variations in pattern: Not only is there a variation in the character of the epithelial cells, but also the arrangement of the epithelial elements in relation to the stroma displays many patterns. The epithelial elements may form masses, strands, or trabeculae forming an alveolar arrangement (Fig. 4). In some cases the epithelium consists of slender anastomosing strands in a plexiform pattern (Fig. 5), while in others a polycystic arrangement is seen; in the latter the cysts usually vary in size, some reaching large proportions (Fig. 6). The adenomatous pattern consists of epithelial cells forming acini (Fig. 7), this type being rarely encountered. In a few tumors studied there was a mixed-tumorlike arrangement of the epithelium in relation to the stroma which is hyalinized in nature.

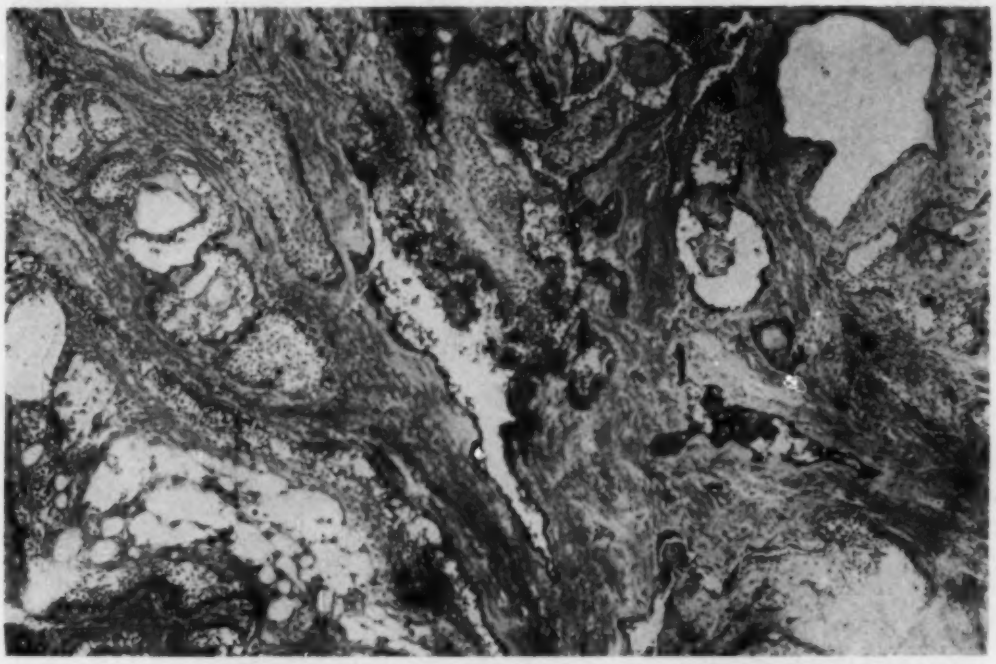
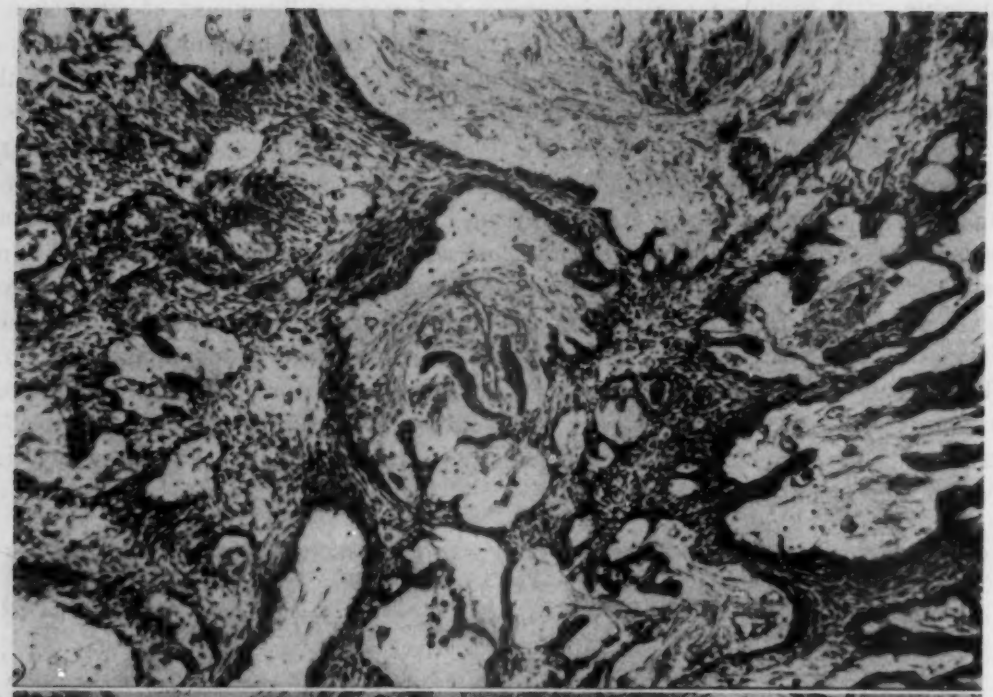


Fig. 4.—Alveolar arrangement of the epithelium in an adamantoblastoma.

Enameloma.—The enameloma is a small tumor, sometimes situated between the roots of two teeth, but more frequently attached at the bifurcation of the roots of molars and premolars and at the cervical margin of single-rooted teeth. Sometimes spoken of as enamel drops or enamel pearls, enamelomas should be differentiated from small supernumerary teeth containing dentine and pulp. Microscopically, the enamel which appears as a space in the decalcified section is covered by a layer of atrophied epithelial cells, occasionally by a layer of cementum.

Fig. 5.



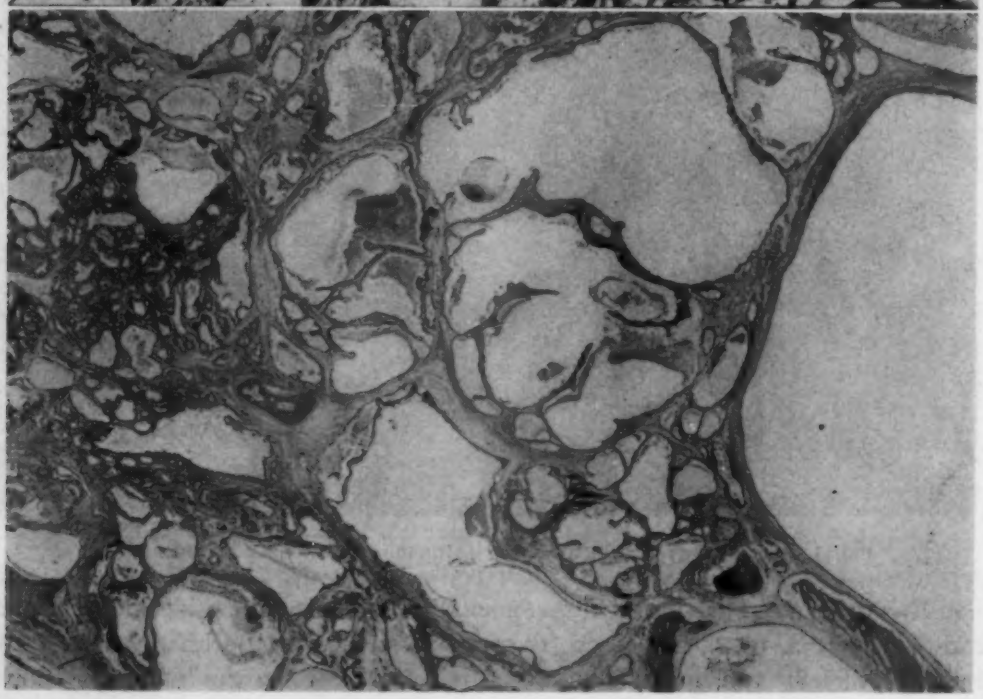


Fig. 6.

Fig. 5.—Plexiform pattern of the epithelium in an adamantoblastoma.
Fig. 6.—Polycystic pattern in an adamantoblastoma.

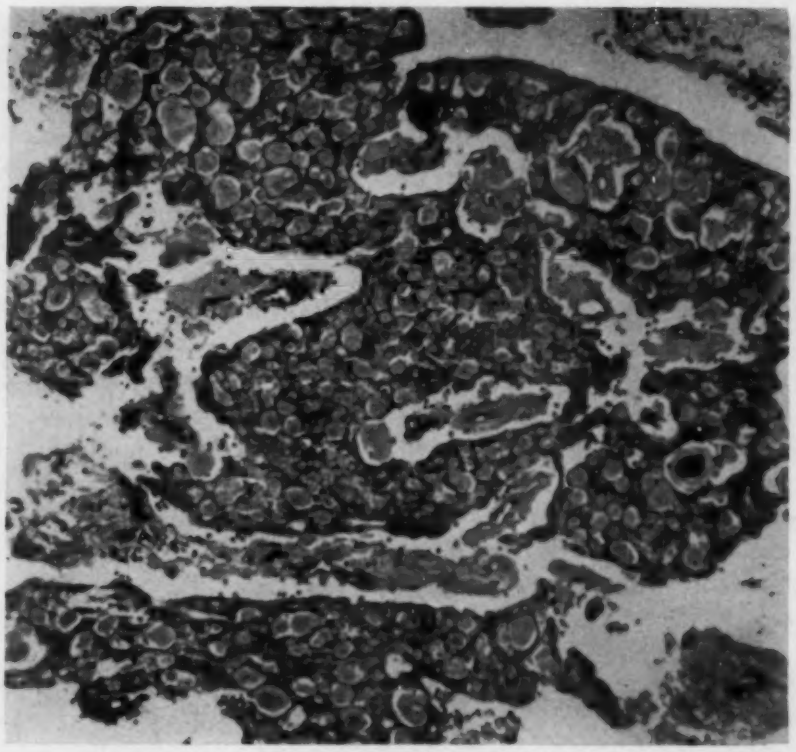


Fig. 7.—Adenomatous arrangement of the epithelium in an adenoadamantoblastoma.

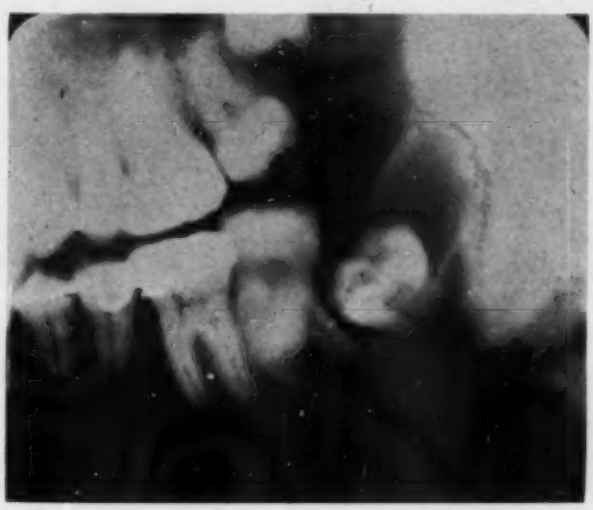


Fig. 8.—X-ray of an odontogenic fibroma.

MESENCHYMAL ODONTOGENIC TUMORS

Odontogenic Fibroma.—This tumor arises from the mesenchymal portion of the tooth germ, that is, from the embryonic tissue of the dental papilla or dental follicle, and, later, from the periodontal membrane. It may, therefore, occur in the coronal or apical regions of the tooth; if in the latter, it may be attached to the tooth and is often mistaken for a granuloma or an odontogenic

cyst. One of us (K. H. T.)[5] has reported a case in which roentgenograms showed the tumor as a cystic lesion enveloping the root of a partially erupted mandibular third molar (Fig. 8). The tumor consisted of a solid mass composed of embryonal connective tissue containing small spindle-shaped cells. No inflammatory infiltration was present. In the fibrous stage it is impossible to determine the outcome of the lesion since it may form either a dentinoma or a cementoma. Dentinoma is usual in the coronal region; cementoma, in the apical region.

Dentinoma.—The dentinoma in the pure form is rare; however, the same process may be found in a few of the odontogenic mixed tumors. The dentinoma, composed of mesodermal tissue, is made up of denticles or islands of irregularly formed dentine in a stroma consisting of connective tissue with enmeshed cells of various shapes, spindle and round being more common. The dentine contains but few tubules, more tortuous than normal, and resembles secondary dentine laid down in the pulp in advance of caries. Some of the dentine encloses living cells to appear as if it had been secreted around odontoblasts. Certain of the interstitial cells closely resemble bone corpuscles, and this type of tissue may be referred to as osteodentine.

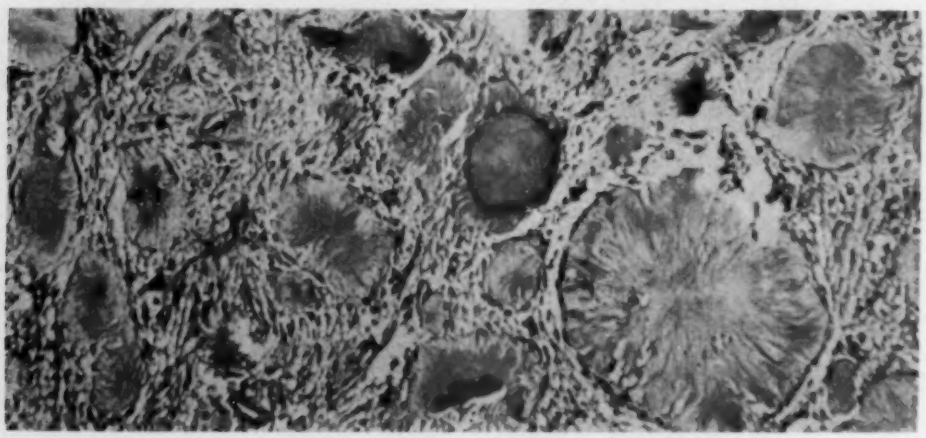


Fig. 9.—Islands of osteodentine in a dentinoma.

In a case of dentinoma studied, the left nostril of a 6-year-old boy was completely obstructed by a firm, movable mass, which had produced slight asymmetry of the face. Deviation of the septum to the right caused difficulty in breathing through the right nostril. The bone over the maxillary sinus could easily be depressed over an area about an inch in diameter. The first dentition was complete. X-ray showed a cystic area above the deciduous central incisor and adjacent to a permanent tooth bud. Microscopic examination of the tissue revealed a rather loose mesenchyma containing numerous round and irregular masses of eosinophilic material recognized as dentine (Fig. 9). This substance was somewhat fibrillar with irregular dentinal tubules; entrapped cells were common (Fig. 10). One part of the tumor was composed exclusively of connective tissue which resembled dental pulp; at the border of the tumor the tissue contained bony spicules which corresponded to the normal bone surrounding the tumor.

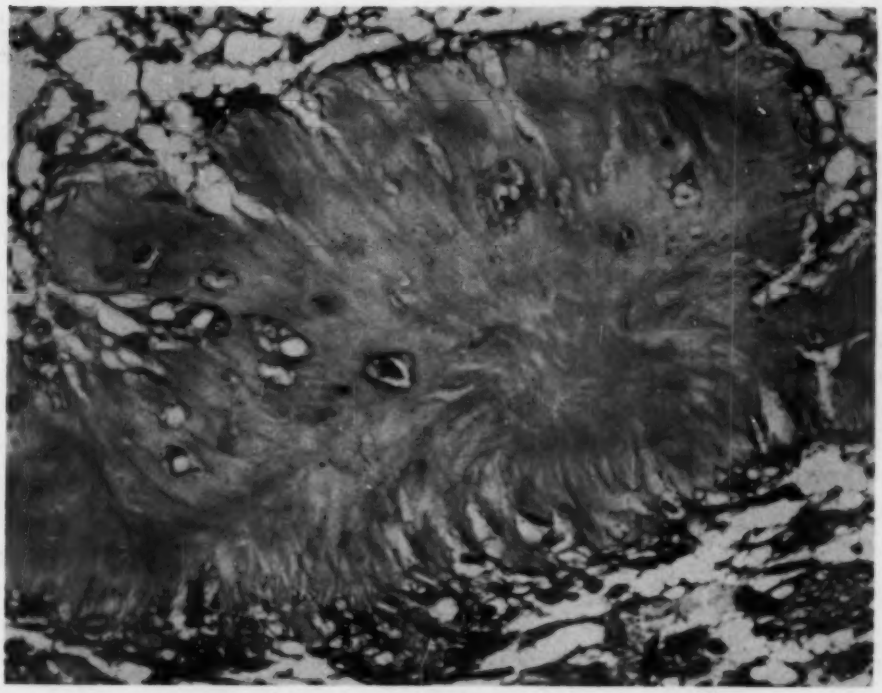


Fig. 10.—High power of Fig. 9, showing character of the osteodentine. This substance is identical with the osteodentine which has been laid down rapidly in advance of caries.

Cementoma.—The cementoma was described by one of us (K. H. T.),[6] who pointed out that it was a secondary formation, the by-product of a soft tissue tumor, the cementoblastoma (odontogenic fibroma). In the growing stage the cellular tissue is predominant, diminishing in proportion as cementum is formed. When the cellular elements have exhausted their activity, they remain as a thin connective tissue capsule around the calcified tumor; thus the histologic picture varies with the stage through which the tumor is passing. The cementoblastoma may produce cementicles which become fused, or trabeculae may be laid down in lamellar fashion. It is difficult to distinguish cementum from bone, especially when there is cementoblastic activity as well as cementoblastic deposition. Cementum, however, has a fibrillar matrix, a more irregular lamination, and fewer inclosed cells than bone.

TUMORS ORIGINATING FROM BOTH ODONTOGENIC EPITHELIUM AND MESENCHYMA

The odontogenic mixed tumor is composed of the epithelial and mesenchymal portions of the tooth germ with the potentialities of those structures to form enamel, dentine, cementum, pulp, and periodontal tissue. Both soft and calcified types are encountered.

Soft Odontoma.—It has been observed that the stroma is often prominent in the solid type of ameloblastoma. Bauer[7] recognized that in cystic ameloblastomas there is usually an insignificant amount of connective tissue, while in the more solid tumors the stroma was greatly increased and played an important role. Thoma[5] described soft odontoma as derived from the mesenchymal

part of the tooth germ which may form from either the embryonic mesenchymal tissue or the dental follicle. He believed that soft odontoma might be classified as central fibroma or fibro-odontoma; however, as dental epithelium was present in many of these tumors, the term fibroadamantinoma might be justified. Nagel[8] pointed out that these soft tumors were easily mistaken for adamantoblastomas.

In odontogenic mixed tumors of the soft type, the epithelium is usually arranged in lobules or strands, the peripheral cells of which are more columnar and resemble prefunctional ameloblasts. The central cells are often squamous- to spindle-shaped, or assume a stellate-reticular form, like that seen in the enamel organ. Sometimes the epithelial strands suggest buds or the bell shape of the early enamel organ. The stroma is abundant, at times embryonal, with stellate fibroblasts as seen in the dentinal papilla and young pulp of a tooth. It may resemble more mature connective tissue in which collagen is deposited, and numerous blood vessels are present. The stroma may become fibromyxomatous. Occasionally the tumor is composed almost entirely of connective tissue with epithelium a minor component.



Fig. 11.—X-ray of a large soft odontogenic mixed tumor in the mandible. Note the multicystic appearance. (Courtesy Army Institute of Pathology, Neg. 78852.)

In a case of *soft odontogenic mixed tumor* studied at the Army Institute of Pathology (A.I.P. Acc. 101059), a painless mass on the face of a man, 29 years old, was firm except for one fluctuant area anterior to the angle of the mandible. Roentgenologic examination revealed a multicystic area (Fig. 11). A large neoplasm had invaded much of the substance of the mandible. The larger portion of the tumor was on the mesial side of the bone, but there are two solid masses within the body of the mandible. The lower border was completely eroded and replaced by a tumor with a bluish surface. It cut with relative ease, was compact, light yellow, gelatinous, interspersed with a few

fine trabeculae, and was surrounded by a thick fibrous capsule (Fig 12). Microscopically, large columns and strands of epithelial cells were embedded in a fibrous matrix. Many epithelial strands were thin and multibranching, winding through contiguous microscopic fields (Fig. 13). In the individual cord the cells in the outermost layer were columnar; those in the center were either squamous or small with clear cytoplasm, giving the tissue a reticulated appearance. Connective tissue of a simple fibrous type was far more abundant in the tumor than epithelial tissue. In many areas, the cells of the stroma were irregularly disposed, their cytoplasm clear, and the nuclei large with prominent nucleoli (Fig. 14).

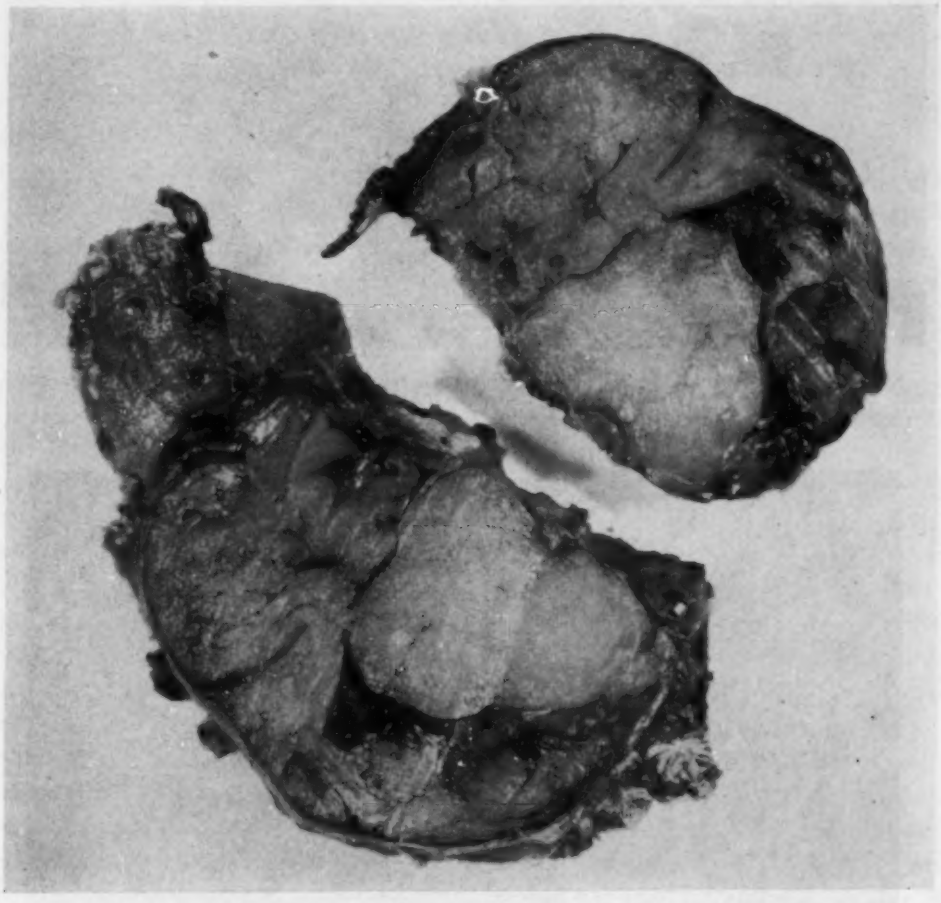


Fig. 12.—Cross section of the gross specimen of a soft odontogenic mixed tumor. The lobulated form can be compared to the x-ray seen in Fig. 11. (Courtesy Army Institute of Pathology, Neg. 81565.)

Soft and calcified odontomas have been divided into two groups: the first characterized by a soft epithelial component, a soft mesenchymal component, and dentine, the second, by a soft epithelial component and enamel, soft mesenchymal component, and both dentine and cementum. The microscopic picture of the first type was similar to that of the soft odontogenic mixed tumor except that dentine was produced. The nests of adamantine tissue were rimmed by dentine which was irregular in structure as is secondary dentine.

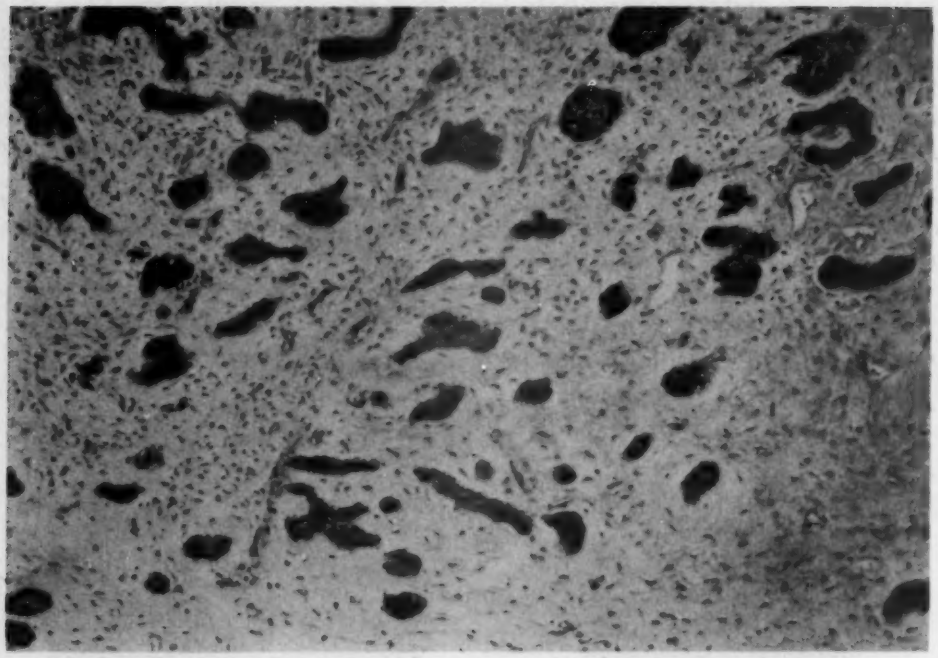


Fig. 13.—Microscopic appearance of the soft odontogenic mixed tumor. The epithelial strands resemble the dental lamina of tooth formation. (×125.) (Courtesy Army Institute of Pathology, Neg. 78576.)

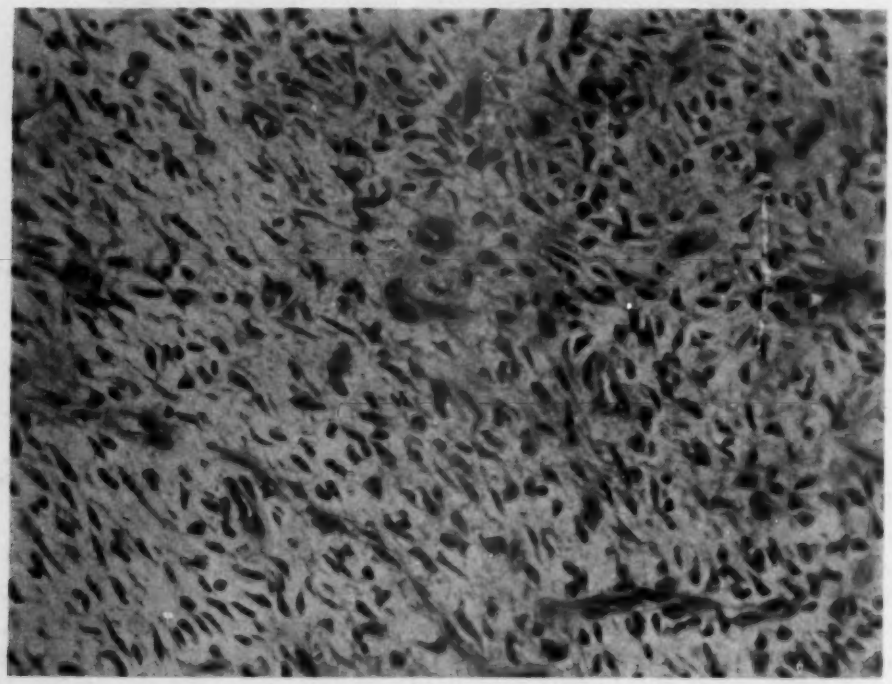


Fig. 14.—The stroma of the soft odontogenic mixed tumor of Figs. 11-13. This structure resembles pulpal tissue (×435.) (Courtesy Army Institute of Pathology, Neg. 78692.)

The dentinal tubules were fewer and were tortuous in contrast to their regularity in normal dentine. This process could be interpreted as a response of the stroma by forming dentine in the presence of adamantine tissue, a reaction commonly observed in odontogenic mixed tumors.

Perhaps the most interesting of all the odontogenic tumors are those in which all possible phases of dental production are seen. In certain tumors, both fully formed and rudimentary teeth or irregular dental structures are found. Areas of pure adamantoblastoma, of pure dentinoma, and of epithelial tumor in conjunction with dentine production may be encountered as well as tooth buds in all stages. These processes are well illustrated in three cases studied. In the first case, an abortive tooth root composed of irregularly formed dentine covered by cementum was adjacent to small rudimentary teeth made up of an epithelial membrane around the enamel spaces. Occasional epithelial sprouts arose from this membrane. In one area the tissue consisted almost entirely of proliferating epithelium which developed in the form of irregular follicles surrounded by a rim of dentine. The epithelium consisted of an outer layer of cylindrical cells and a central portion of squamous cells which tended to form pseudo pearls. In places, the rim of dentine was absent; in others, dentine formation was beginning. In the second case, the processes were essentially the same, but in addition there were areas of dentine laid down in denticle fashion, epithelium undergoing necrobiosis, and epithelium arranged in acini filled with a colloidlike material. In the third case, filling the cavity of a cyst was a mass of irregular dental structures in a connective tissue stroma. Between these structures were concentric masses of necrobiotic epithelial cells in which focal areas of calcification were noted. Scattered through the collections of dentine and necrobiotic epithelium were aggregates of adamantine cells, in some areas solidly packed, in others assuming a cystic pattern. In the capsule of the cyst, lobules of adamantine tissue and scattered ghostlike epithelial cells were intermingled. Of greatest interest was the formation of a small tooth bud in the capsule. It was composed of layers of epithelium, dentine, an irregular odontoblastic layer, and the dental papilla. The epithelium, however, had not differentiated into ameloblasts although a considerable layer of dentine had been laid down. These cases suggest the inductive effects of one tissue upon another. Epithelial lobules seem to evoke the potentiality of the adjacent stroma, causing dentine to be laid down. Dentine was formed despite the lack of an ameloblastic layer and in several instances in the absence of epithelium.

The following are histories of four cases of odontogenic mixed tumor studied:

Case 1.—A white woman, 35 years old, had had a painless swelling of the left side of the face for one and one-half years (Fig. 15). A firm mass over the left ramus of the mandible was shown by roentgenologic examination to contain a large cystic cavity involving the entire left ramus and part of the mandible, and to encase the crown of a molar tooth. Above the tooth were numerous, fused, radiopaque masses (Fig. 16). The material removed at operation consisted of an irregular, reddish pink, smooth sheet of membranous tissue

and attached fibrous tissue in which several firm, white, smooth, encapsulated nodules were embedded. At one corner of the sheet was an irregular reddish pink, mucosa-covered mass of firm tissue studded with many pearly white, round, smooth calcified nodules. In the center was an abortive tooth. Microscopically,



Fig. 15.



Fig. 16.

Fig. 15.—Photograph of patient with odontogenic mixed tumor.

Fig. 16.—X-ray of odontogenic mixed tumor. Note the numerous fused radiopaque masses above the embedded tooth.

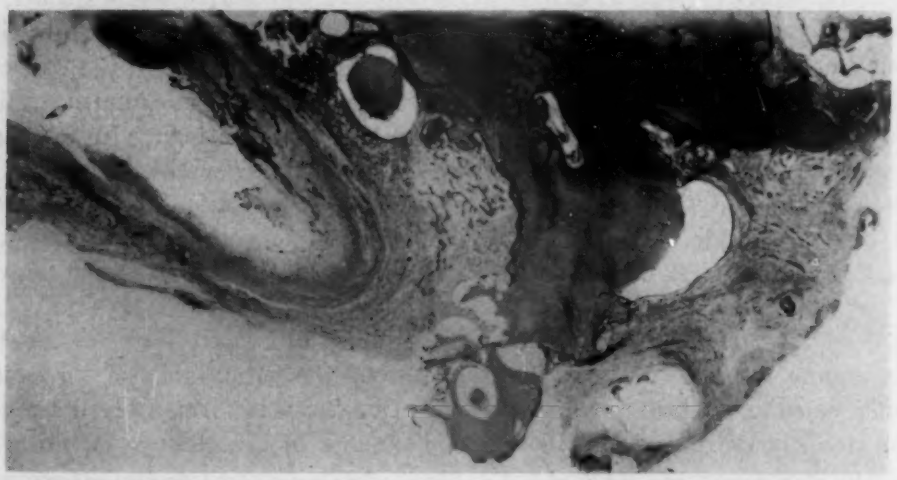


Fig. 17.—This is a photomicrograph of an abortive tooth and irregular dental formation in an odontogenic mixed tumor. A cyst sac is seen at the left, the capsule lined by epithelium of adamantine nature.

the large calcified mass (described as the abortive tooth root) was made up of irregularly formed dentine and covered by projections of cementum; close by were small rudimentary teeth (Fig. 17). Epithelial membranes surrounded spaces which represented the enamel dissolved during decalcification. Some of the more immature enamel was still attached to a core of dentine which in some instances contained a central pulp canal. Connective tissue surrounded

some of the epithelial membrane, and, occasionally, cementum extended from the tooth to join the connective tissue. Lining a cystic space and extending into the connective tissue were masses of small, deeply staining epithelioid cells which had a tendency to form small acini. At one place, epithelial structures, rimmed by dentinoid, had proliferated into the adjoining tissue (Fig. 18); at another,

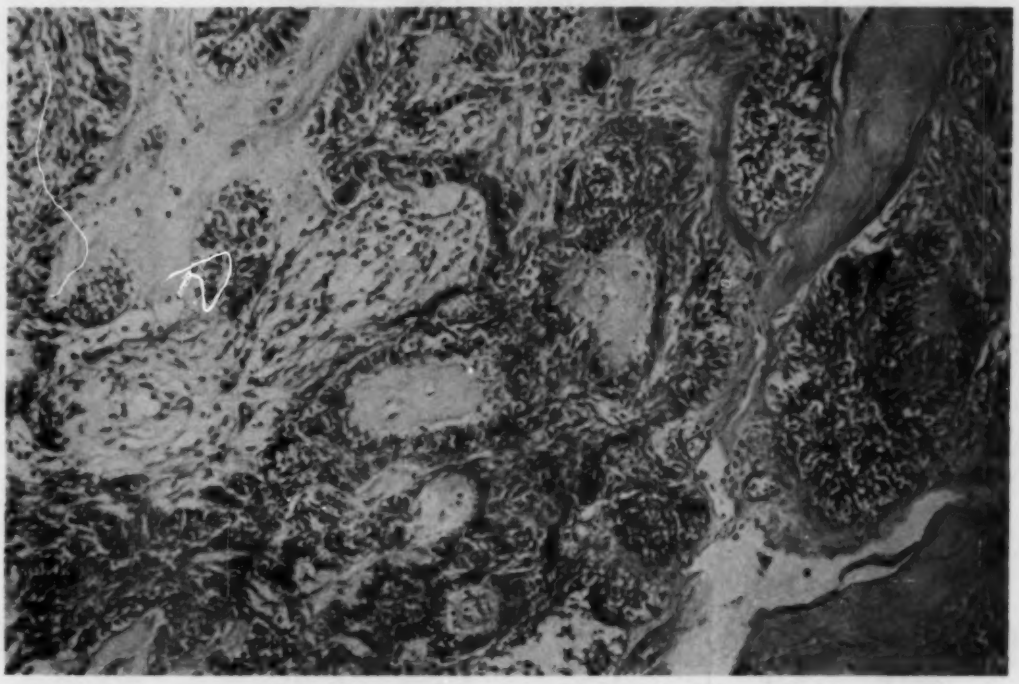


Fig. 18.—Odontogenic mixed tumor. Note the epithelium rimmed by a dentinoid structure.

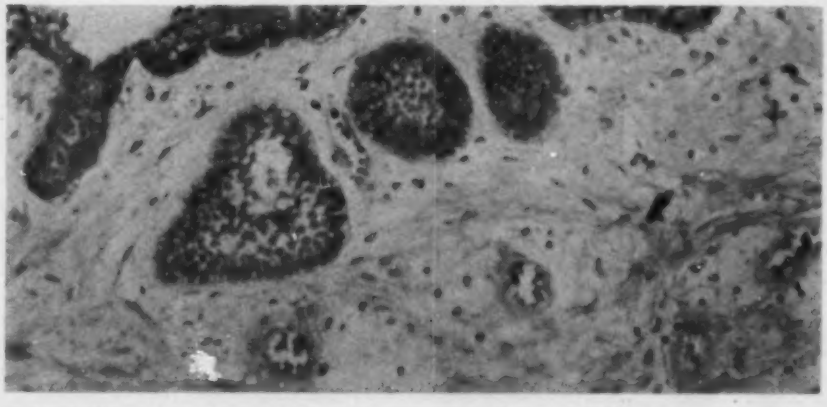


Fig. 19.—This is a photomicrograph of lobules of epithelial cells in the connective tissue capsule seen in Fig. 17. The largest lobule is undergoing central cystic degeneration.

proliferation of epithelium had produced irregular papillary structures surrounded by connective tissue. The papilla was made up of an outer layer of cylindrical cells and contained a variety of cells, some of which had undergone squamous metaplasia and had formed pseudo pearls. Rimming many of these epithelial structures was an eosinophilic material which consisted of irregular

tubules and a few cellular inclusions in a somewhat fibrillar material. This material was identified as dentine. Scattered through the connective tissue capsule were lobules of adamantine cells, many groups of which showed central cystic degeneration (Fig. 19).

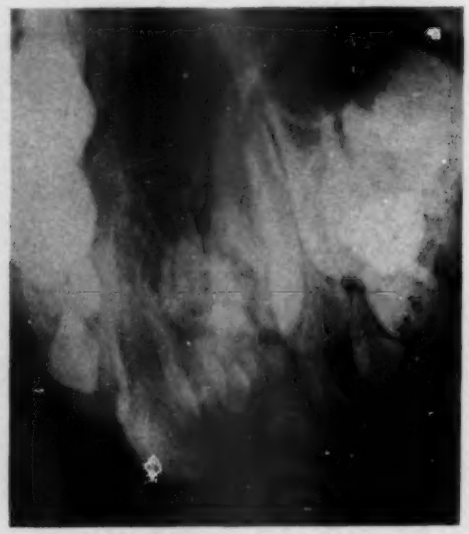


Fig. 20.—X-ray of odontogenic mixed tumor in an 8-year-old boy.



Fig. 21.—X-ray of maxilla taken six years before x-ray in Fig. 20. A small cystic area is seen; this was not removed, suggesting that the odontogenic mixed tumor developed in this cystic area.

Case 2.—A boy, 8 years old, had a diastema between the maxillary central incisors and a hypertrophied labial frenum. On the right were the two permanent incisors, on the left the two retained deciduous ones. Roentgenologic examination disclosed a calcified mass (Fig. 20), distal to which were three

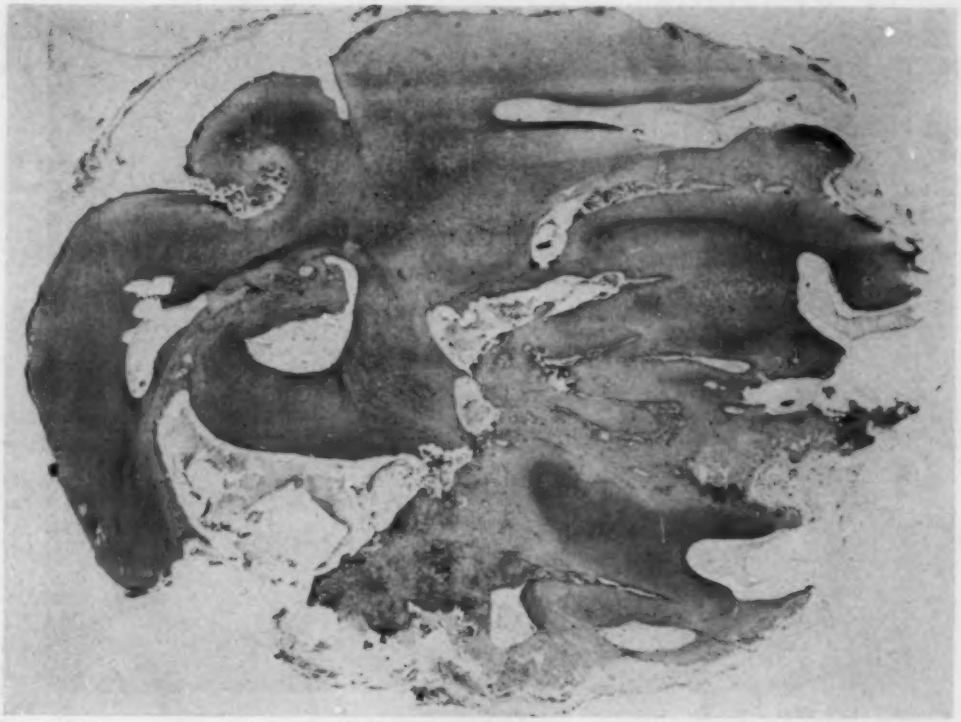


Fig. 22.—Low-power microscopic appearance of the odontogenic mixed tumor in Fig. 20.

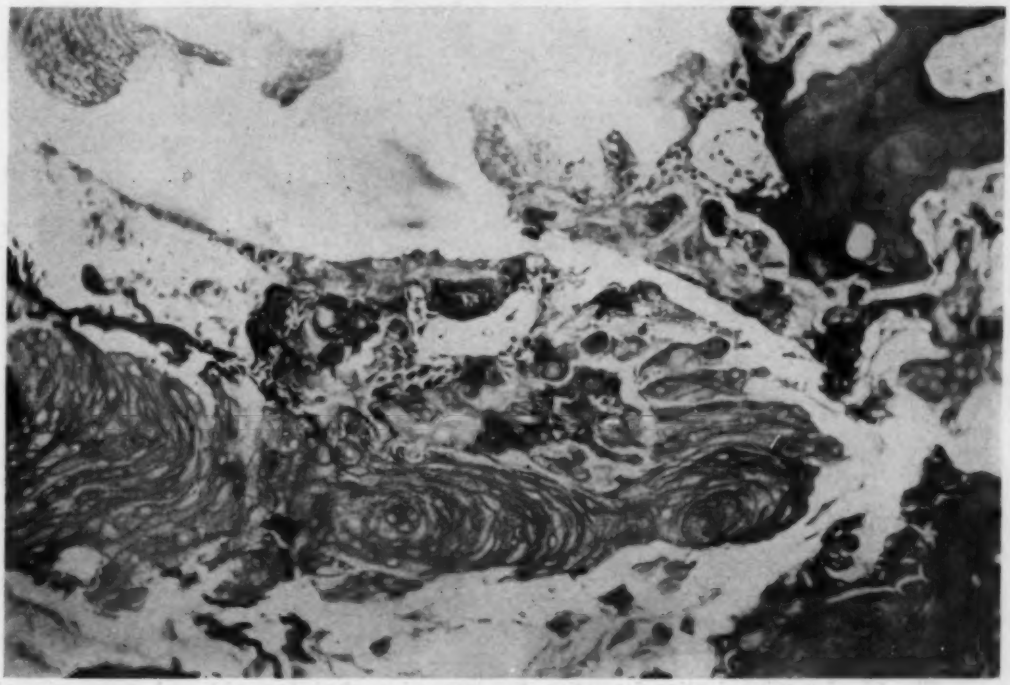


Fig. 23.—Ghostlike epithelial cells undergoing calcification in the odontogenic mixed tumor, the x-ray of which is seen in Fig. 21.

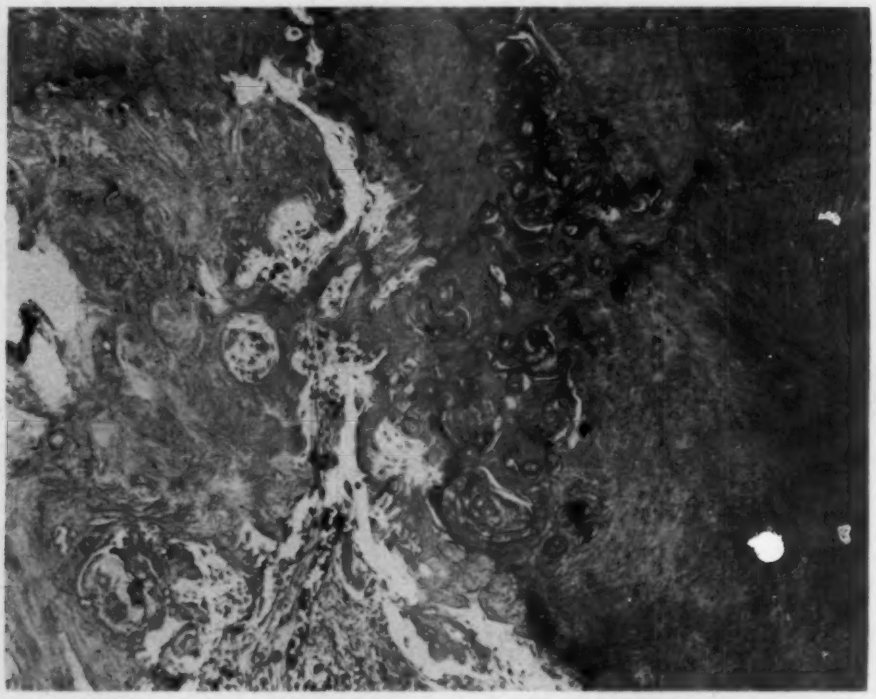


Fig. 24.—Attached to dentine are masses of ghost cells of epithelium in the odontogenic mixed tumor, the x-ray of which is seen in Fig. 21.

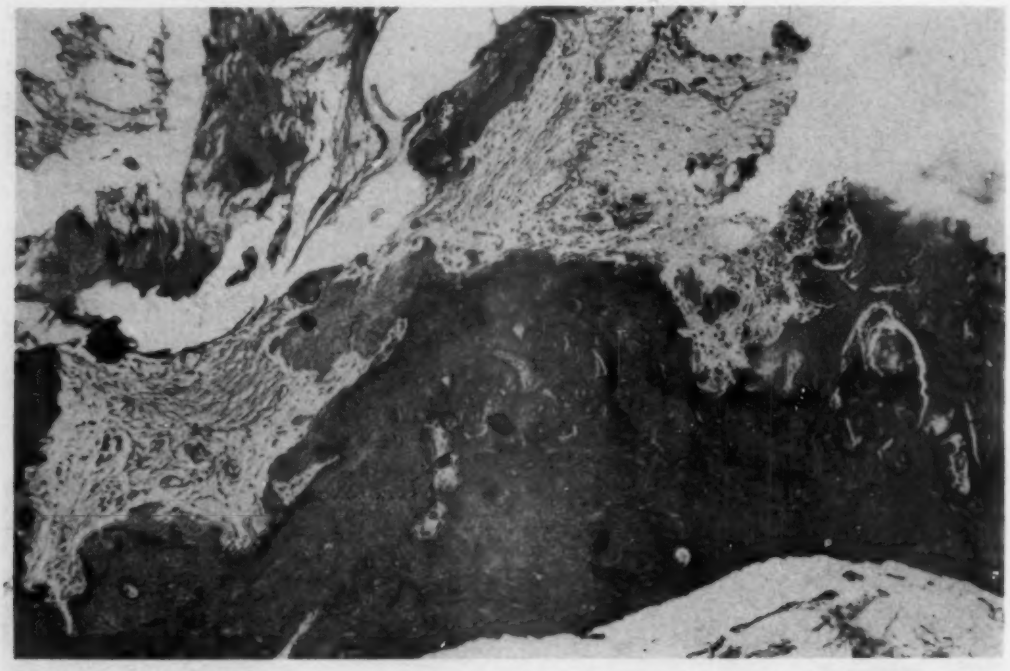


Fig. 25.—Irregular dentine formation (lower portion of photograph) and ghost cells of epithelium (upper left portion) in the odontogenic mixed tumor, the x-ray of which is seen in Fig. 21.

unerupted teeth. Six years before, the left central deciduous incisor failed to erupt normally and a small cystic lesion was noted in the roentgenograms but was not removed (Fig. 21). This history suggested an odontoma which had formed in the cystic area and which probably had at first been soft but later had expanded to form calcified dental structures. Microscopic examination showed that the mass was composed of the various elements of the tooth, arranged in the irregular pattern consistent with odontoma (Fig. 22), and that it was encapsulated by fragmentary connective tissue which in one area contained adamantine epithelium arranged in acini filled with a mucoid substance.



Fig. 26.—Remnants of an ameloblastic layer over remnants of enamel. Note the adjacent flattened epithelium the pattern of which has the tendency toward acini.

Lying in dentine or enamel spaces were collections of ghost epithelial cells which had undergone calcification (Fig. 23). In some areas there was evidence of necrobiosis of the epithelium preceding calcification. Attached to the normal dentine were masses of irregularly formed osteodentine around ghostlike epithelial cells (Figs. 24 and 25). At one place, remnants of an ameloblastic layer lay over an enamel space; this layer was adjacent to flattened epithelium which inclined toward an acinar arrangement (Fig. 26).

Case 3.—A boy, 16 years of age, noticed a swelling under his upper lip which had been growing for several months, and tenderness of the left maxillary lateral and central incisors. Bulging of the labial surface of the alveolar process prevented the lips from closing. The swelling was firm but crepitant. The roentgenogram disclosed a cystic area extending from the alveolar process of the anterior part of the maxilla into the palate. In the center was a radiopaque mass, irregular and unevenly calcified (Fig. 27). Microscopically, the cyst was composed of vascular fibrous connective tissue lined by stratified squamous epithelium. Springing from one side of the cyst wall and nearly filling the cavity was a mass of toothlike material in connective tissue stroma (Fig. 28).



Fig. 27.—X-ray of an odontogenic mixed tumor. A large cystic area is seen extending from the alveolar process to the anterior part of the maxilla into the palate. In the center is a large radiopaque mass, irregular and unevenly calcified.

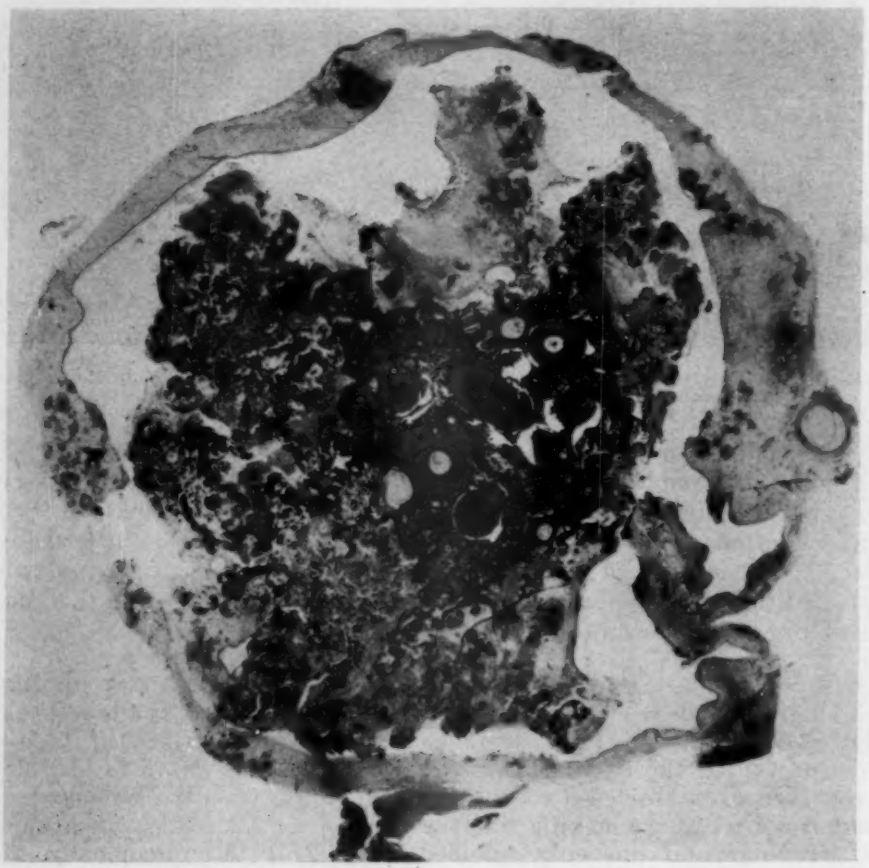


Fig. 28.—Photomicrograph of the cyst composed of fibrous connective tissue lined by stratified squamous epithelium. Arising from one side of the capsule and nearly filling the cavity is a mass of irregularly formed dental structures in a connective tissue stroma. Note the small tooth-bud formation in the capsule on the right.

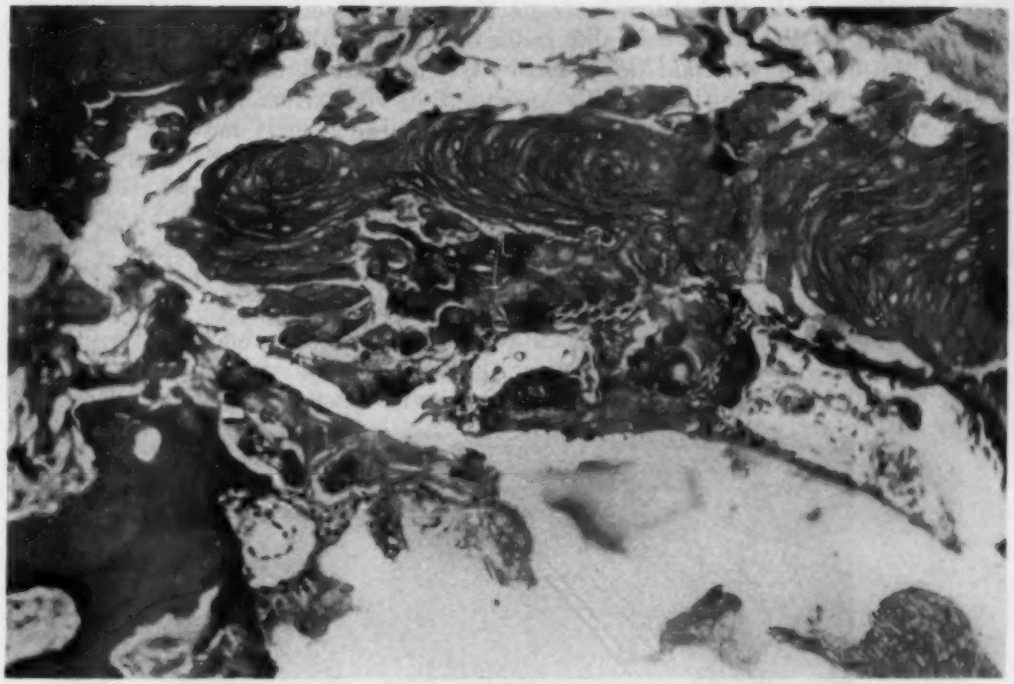


Fig. 29.—Concentric masses of necrobiotic epithelial cells found in between the hard structures seen in low power in Fig. 28.

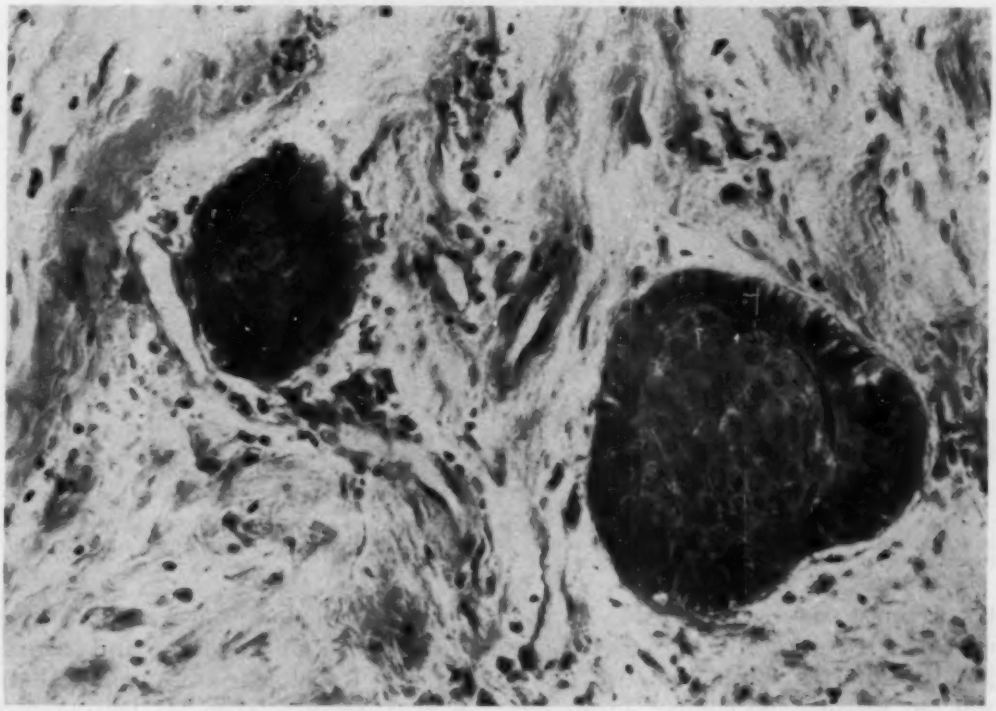


Fig. 30.—Lobules of adamantine epithelium seen in the capsule of an odontogenic mixed tumor.

Two or three of the structures were nearly normal teeth on a small scale, the others were smaller, less well differentiated, and without pulp centers. Although the dentine was laid down normally, the enamel was not arranged correctly. Necrobiotic epithelial cells arranged in concentric masses, resembling the spider cells of an adamantoblastoma, were found largely between the small, poorly differentiated teeth (Fig. 29). In some of the masses, focal areas of calcification were seen. Aggregations of adamantine epithelial cells were scattered among collections of dentine and epithelial ghost cells. Most of this adamantine tissue was solidly packed but in some areas it assumed a cystic pattern, the spaces being filled with a colloidlike material. Here and there, dentine had been laid down haphazardly. In the capsule were accumulations of the same necrobiotic epithelial cells as in the lumen, also lobules of epithelial cells of distinctly adamantine nature (Fig. 30). The peripheral cells were either cuboidal or columnar, resembling the prefunctional ameloblast (Fig. 31).



Fig. 31.—Medium-power photomicrograph of the tooth-bud formation seen in Fig. 28. It is composed of epithelium, dentine and dental papilla, but the epithelium does not suggest functional ameloblasts. There is no attempt at enamel formation.

Of greatest interest was the formation of a small tooth bud in the cyst capsule. Structurally it was composed of epithelium, dentine, and dental papilla, but the epithelium did not suggest functional ameloblasts and there was no attempt at enamel formation. A considerable layer of dentine was evident, although the odontoblastic layer was not regular or even fully developed; the dental papilla was characteristic.

CASE 4.—A swelling on the right side of the face of a boy, 15 years old, was confined to the ascending ramus. Gradual increase in size and slight pain noticeable for nine months were attributed to a blow. On the right side of the mandible the second and third molars were missing. The roentgenogram showed a two-compartment cyst: the first, below the roots of the first molar, contained a tooth displaced to the inferior border; the second, which occupied the entire ramus and expanded its anterior border, contained a mass of round calcified particles (Fig. 32). Microscopically, the cyst capsule (Fig. 33), was composed of dense connective tissue lined by adamantine epithelium, some of which was necrobiotic (Fig. 34). The adamantine cells were, for the most part, packed closely and their nuclei stained deeply. Attached to the adamantine tissue and situated in the capsule were many embryonal tooth organs composed of a layer of ameloblasts surrounding a dental papilla (Fig. 35). An attempt to form an odontoblastic layer was noted. Between the tooth organs was a stellate reticular tissue, and in some of them enamel and dentine were formed (Fig. 33).

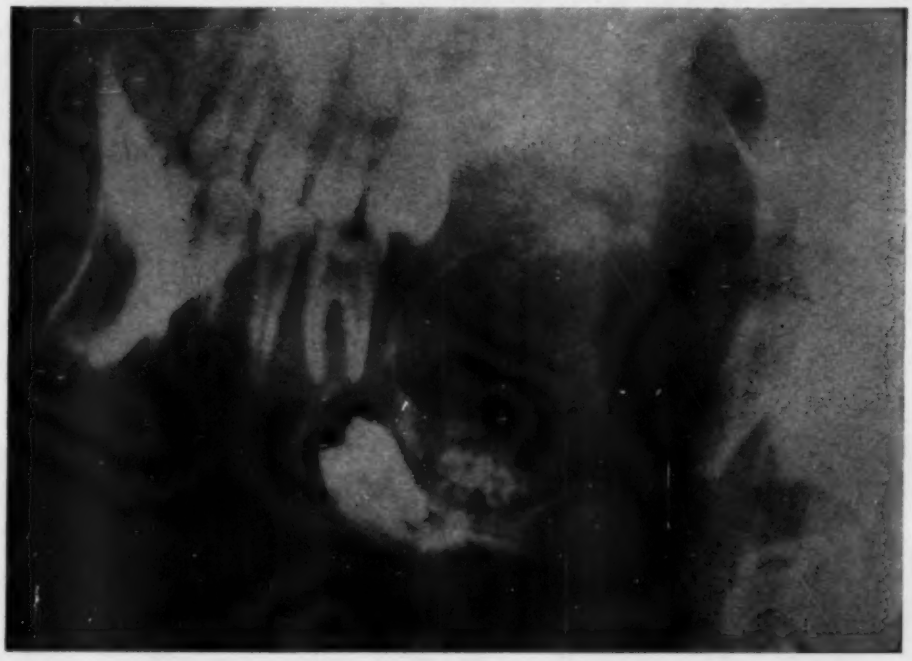


Fig. 32.—X-ray of an odontogenic mixed tumor showing a two-compartment cyst. The first is below the roots of the first molar, and contains a tooth displaced to the inferior border; the second occupies the entire ramus and has in it a mass of round calcified bodies.

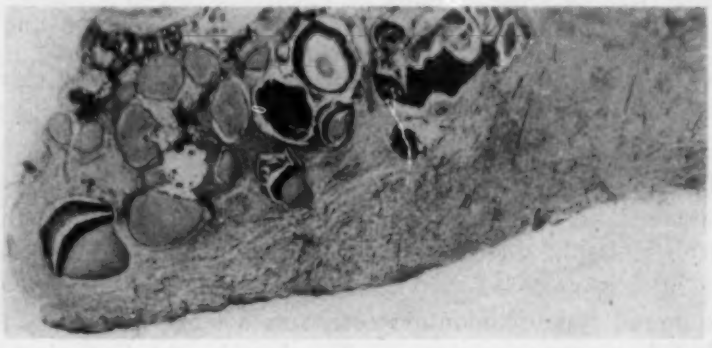


Fig. 33.—Low-power photomicrograph of the capsule of case in x-ray in Fig. 32. Irregular dental structures are seen, some of which are calcified.

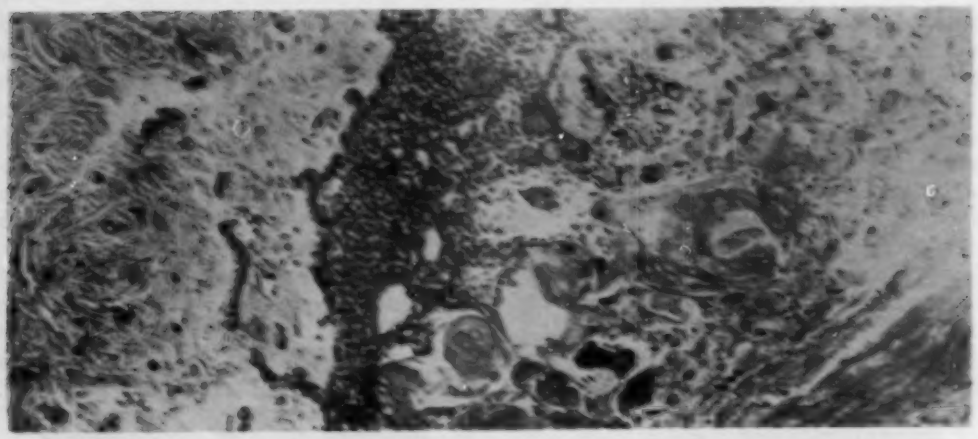


Fig. 34.—High-power photograph of the capsule lining of Fig. 33. The epithelium is of adamantine nature.

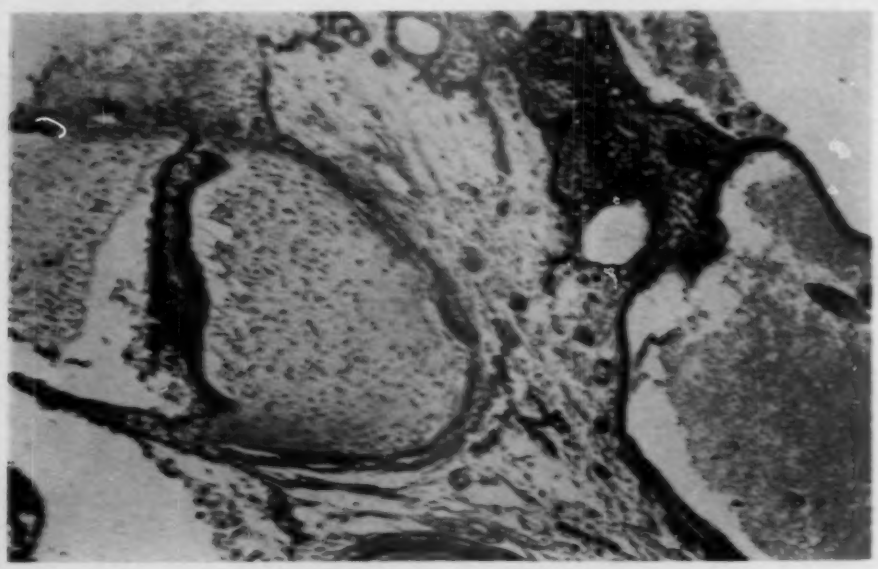


Fig. 35.—High-power photomicrograph of case seen in Figs. 32-34. Seen are a cyst structure (on right), adamantine epithelium (upper right), and embryonal dental formation (on left).

Attached to the functional ameloblastic layer was neoplastic adamantine tissue (Fig. 35), the cells of which tended to be arranged around small cysts. In several places in the capsule, masses of irregularly formed dentine with sparse and irregular tubules were laid down; cellular inclusions were found in the dentine. In one area there was an irregular formation of tooth substance as in a complex odontoma.

Completely formed odontomas include: (1) compound odontomas which are made up of a large number of more or less rudimentary teeth; and (2) complex odontomas in which the calcified structures bear no resemblance to the anatomic arrangement of the dental tissue (Figs. 36 and 37). These tumors are encompassed by capsules.

The compound type of odontoma contains a large number of teeth and is usually included in a cyst membrane. The dental epithelium, instead of form-

ing the normal enamel organ, produces many small enamel organs which develop into tooth germs and give rise to small teeth of varying size and shape. Several adjoining tooth germs may take part in the process and produce some normal, and many deformed, teeth. Microscopically, the structure appears



Fig. 36.—Photomicrograph of complex odontogenic mixed tumor.

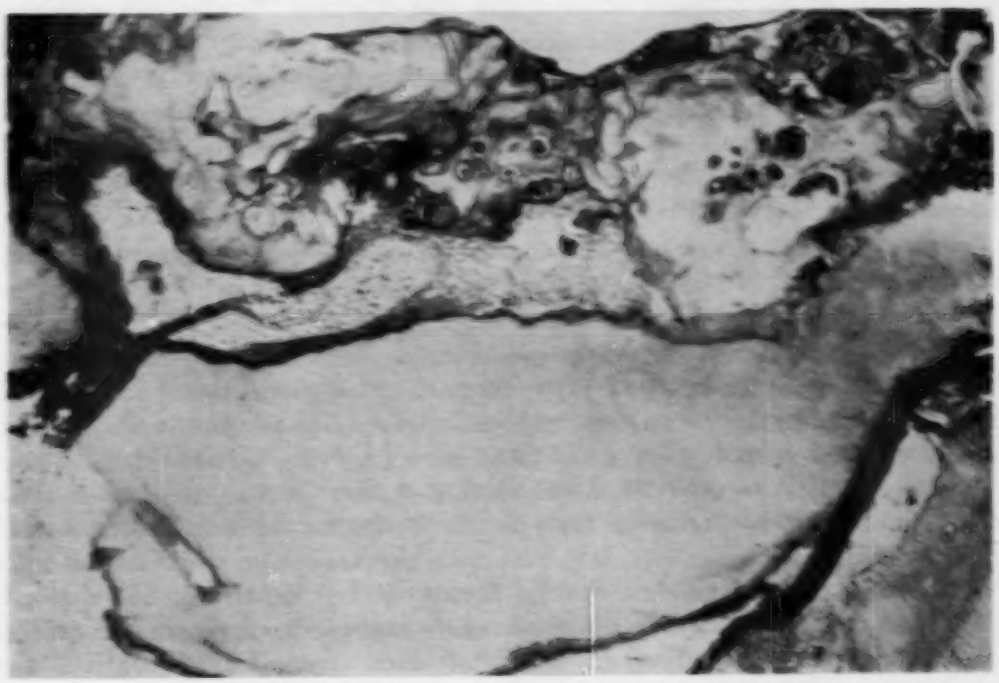


Fig. 37.—High-power photomicrograph of the irregular dental structure seen in case in Fig. 36.

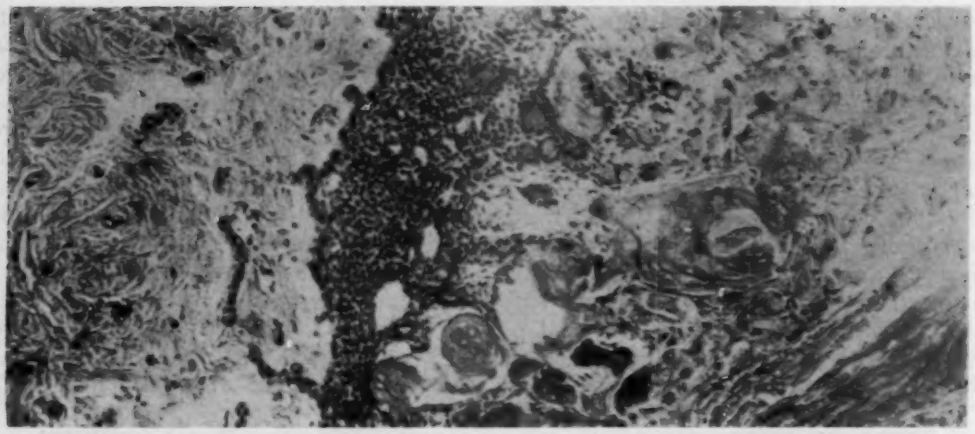


Fig. 34.—High-power photograph of the capsule lining of Fig. 33. The epithelium is of adamantine nature.

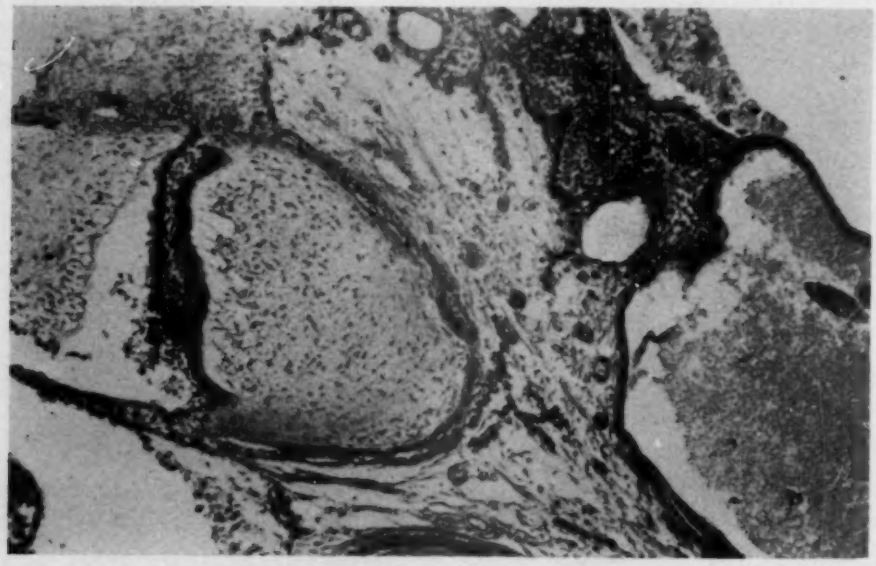


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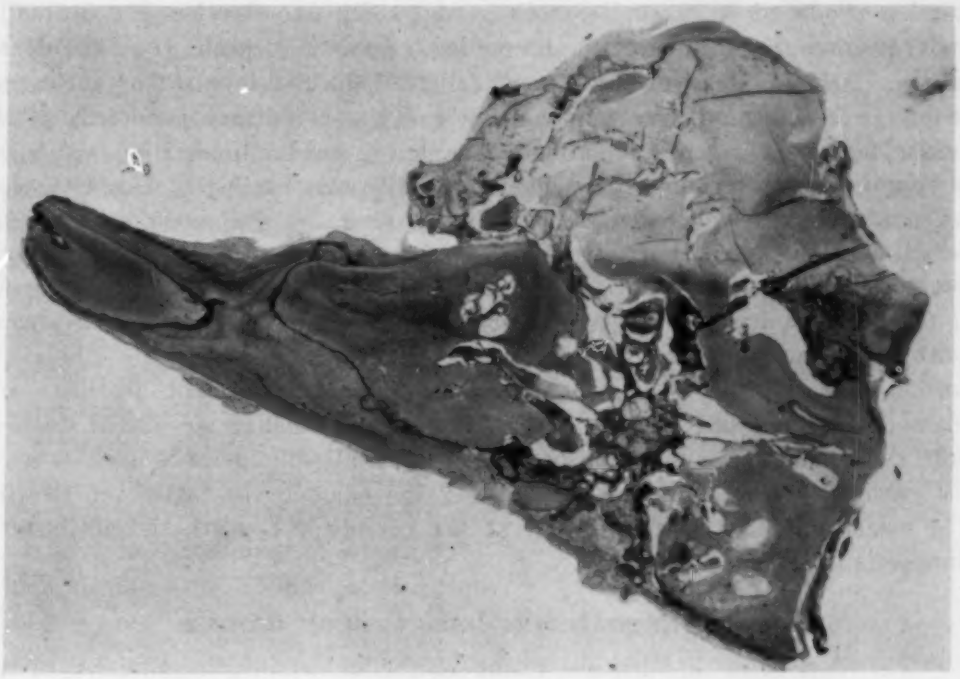


Fig. 36.—Photomicrograph of complex odontogenic mixed tumor.

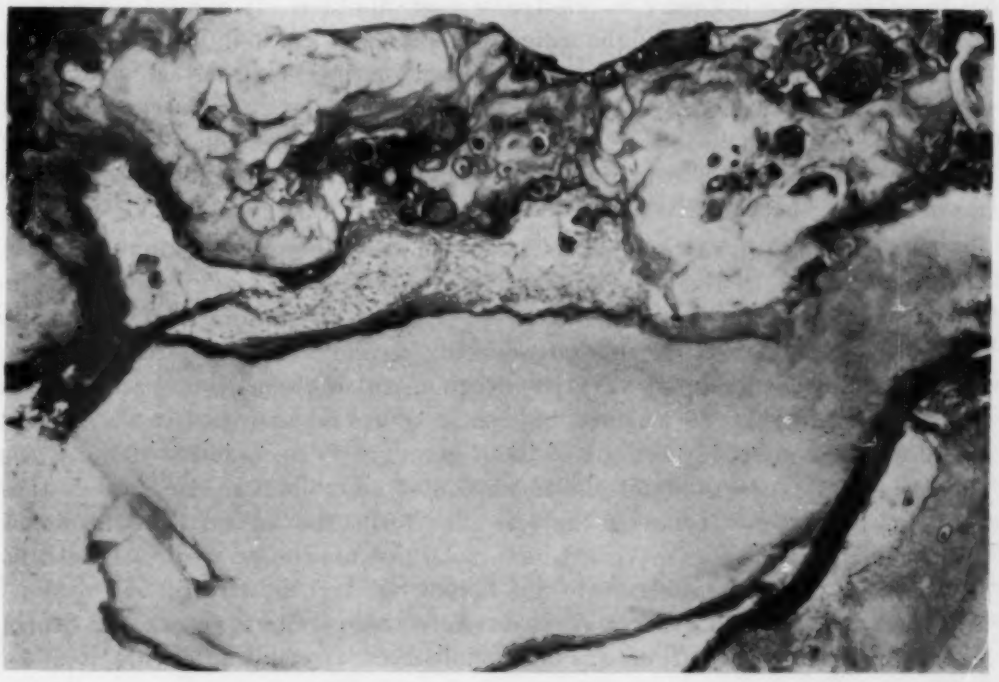


Fig. 37.—High-power photomicrograph of the irregular dental structure seen in case in Fig. 36.

normal, although the teeth may be irregular and dwarfed and may be attached to one another by fibrous connective tissue, cementum, or bone.

In complex odontoma, the arrangement of the tissue is not regular and toothlike, since the tissue is in different stages of development. This tumor may be produced by a single tooth germ, which has developed abnormally, derived either from a normal tooth or a supernumerary sprout from the dental lamina. Schour, Massler, and Greep[9] followed the development of a complex odontoma in a group of rats in which there was hereditary anodontia of the incisor tooth. A complex odontoma is a hard, solid, rounded or oval mass, surrounded by a capsule from which it usually can be shelled out with ease. Its surface may be covered with cementum having scattered nodules of enamel. Sometimes unerupted, normally formed teeth may be fused with the tumor. Microscopically, great variations are found in structure and in the proportion of the different tooth elements. In some, an arrangement like that of normal tooth formation is interrupted by irregularly formed tissue. In others, abnormal arrangement of tooth substance is the predominant feature, with enamel, dentine, and cementum distributed in lamellar or radial fashion. Soft tissue, such as enamel epithelium and dental pulp, may be seen between the calcified layers, the former adjoining the enamel, the latter the dentine. The cementum may be found only at the periphery beneath the epitheliated fibrous capsule.

INDUCTIVE EFFECTS OF ONE TISSUE ON ANOTHER

The deposition of dentine about epithelial lobules, necrobiosis of the epithelium with resultant calcification, and irregular dental patterns bring up the question of the influence of one tissue upon another. In the intimate tissue mixtures of these tumors, neighboring tissues seem to exert inductive influences upon one another, comparable to those known in embryonic development or in mixed tissue cultures. Structural features indicate that odontogenic mixed tumors are not a fortuitous jumble of independent ingredients of a potpourri of tissues, but that the form they take results from the response of one tissue to another.

The relationship of the ameloblast to the odontoblast is of interest as well as of importance in the determination of the type of tissue formed and the pattern developed in odontogenic mixed tumors. Huggins, McCarroll, and Dahlberg,[10] by the method of transplanting developing dental tissues, found that enamel was deposited only on dentine, although dentine could be laid down independently of enamel but only when odontoblasts were present. Glasstone,[11] by growing portions of tooth germs in vitro, found that the ameloblastic layer was essential for the formation of odontoblasts. Because of these findings, Sprawson[12] believed that, in the formation of adamantoblastomas, enamel is not formed. The cells which make up this tumor are basal cells, not ameloblasts, since odontoblasts do not become differentiated. In our study, it appeared that enamel was not formed where there was an absence of dentine and that dentine formation was independent of ameloblasts. This is shown

in Figs. 10 and 30 and also in like areas in the odontogenic mixed tumors. It is, however, possible that dentine production was initiated by fibroblastic activity rather than odontoblastic, with the resultant bonelike dentine seen in these tumors.

The dentinoma has been considered an odontoma, the predominant tissue being dentine. However, in all dentinomas previously described, enamel and cementum were present. A dentinoma consisting purely of dentine and stroma has not hitherto been reported. This tumor arises from the odontogenic fibroma which may differentiate into either cementoma or dentinoma. In one tumor studied, both tissues were present, which suggested their common origin. The structure of the dentinoma demonstrates that dentine may be produced despite the lack of odontogenic epithelium.

The stroma seen in the adamantoblastoma is easily distinguished from the connective tissue observed in the soft odontogenic mixed tumor. It is usually minimal in quantity and does not play an important role in the tumor. In the odontogenic mixed tumor, however, the connective tissue is usually predominant, the epithelium scanty. In these tumors there is a tendency for the production of dentine in apposition to the epithelium. Surrounding many of the epithelial nests is a clear zone, around others a rimming of dentine of the osteodentine type with no odontoblastic layer. Like bone, the dentine has entrapped cells which appear to be of fibroblastic origin.

One must regard the tumors which arise from epithelium and connective tissue as mixed tumors. In further proof are the cases reported as adamantinosarcomas. Krompecher[13] reported one of the first of these. A soft, primary tumor was found in a 13-year-old boy. The histologic picture was that of spindle-cell or round-cell sarcoma, the epithelial part resembling the ordinary type of adamantoblastoma. Wigdortschink[14] described a similar tumor in a boy, 9 years old, who had a lesion in the mandible which appeared to be a sharply circumscribed cyst. Besides highly differentiated adamantine tissue, it contained very cellular connective tissue of a fibrosarcomatous character.

The odontogenic mixed tumor may arise as either a soft or a calcified tumor, the epithelial and mesenchymal elements varying as to differentiation and proportionate quantity. The soft odontogenic mixed tumor is composed of epithelial and mesenchymal elements, and, in those observed, the connective tissue predominated. Morphologically, it resembles the dental pulp, the cells being stellate. The hard, calcified, odontogenic mixed tumor has been frequently described in the literature and has been termed odontoma. The frequent occurrence of soft epithelial polyhedral and squamous cells as well as adenomatous structures in this hard tumor was noted; in many instances these cells were of adamantine nature like the cells of pure adamantoblastoma. Occasionally, irregular calcospherites were seen in the epithelial nests. Thus the combination of soft and calcified odontogenic mixed tumor produces many bizarre pictures, because of the varying proportions and arrangements of the epithelial and mesodermal elements.

LIMITATION OF GROWTH

From the study of this group of tumors, it appears that differentiation to a variety of forms may take place, depending to a large extent on the development of the epithelium and mesoderm at the time the neoplasm begins to form. It seems that the uncalcified tumors undergo a quantitative growth with but little qualitative alteration, while the calcified ones undergo a quantitative and a qualitative change, that is, their growth is one with differentiation. Where evolution is chiefly quantitative, the possibility of uncontrolled growth is far greater than when the evolution is chiefly qualitative, for differentiation tends to lead to the development of the final form. Thus we find that the adamantoblastoma is inclined to grow large and be invasive, while the odontoma appears to grow only to a limited size. The odontogenic mixed tumor has the developmental potentialities of both adamantoblastoma and odontoma, a fact that has received further proof from this series of cases.

SUMMARY

The odontogenic tumors are classified into three groups: Epithelial, mesenchymal, and mixed. The dentinoma, a pure mesenchymal tumor, is composed of connective tissue in which denticles or islands of irregularly formed dentine are present. The odontogenic mixed tumors consist of epithelial and mesodermal elements which are in combination in various proportions and arrangements. Three types are recognized: Soft, soft and calcified, and calcified. The soft type has been differentiated from the solid adamantoblastoma.

There is evidence of the inductive influences of one tissue on another in the odontogenic mixed tumors. It is noted that epithelium in these tumors seems to stimulate dentine formation, but that the presence of epithelium is not necessary for the production of dentine. Also, dentine is formed in the presence of epithelial cells not differentiated into ameloblasts. Neoplastic adamantine tissue and enamel-forming ameloblasts have been distinguished. The presence of these two types accounts, in part, for the formation of the soft and calcified odontogenic mixed tumors.

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ABSORBABLE GELATIN SPONGE AND THROMBIN IN ORAL SURGERY

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NORMAL hemostasis and the formation of a blood clot are of primary concern in oral surgery. In even simple extractions rapid clotting helps to prevent infection of a jaw through an open socket and initiates the process of repair and healing. Absorbable materials which aid hemostasis and can be used to accelerate healing are a boon to surgery. The healing process is a corollary to the physiologic sequence of blood clotting. In the healing of large cavities, such as are occasioned by the removal of cysts or impacted lower third molars, repair is often complicated and delayed by the breakdown of the initially formed clot. Because the organization of large clots is uncertain, operators often resort to the use of drains, dressings, and wicks. All of these devices, used thus as "space obliterators," retard healing and necessitate increased postoperative care. Fibrin foam has recently been offered by various authors[1-4] as the solution to hemostasis and space obliteration in oral surgery, and it does the work well. It is, however, very difficult to obtain, being a human blood fraction, and it is also somewhat troublesome to handle. For these two reasons we have worked with Gelfoam and thrombin, a commercial product readily available, nonfriable and hence easy to use.

Gelfoam* is the proprietary name of an absorbable gelatin sponge originally developed for neurosurgical work by Correll and Wise.[5, 6] The sponge, soaked with beef thrombin, acts as an effective hemostatic and has the advantage of being completely tolerable to human tissue and absorbable. In experiments conducted by Light and Prentice,[7, 8] Pilcher and Meacham,[9] and others, it has been shown that tissues tolerate Gelfoam, just as they do fibrin foam, with no appreciable reaction and that there is complete absorption of both these products. Whereas human thrombin is used in conjunction with fibrin foam, the much more easily supplied beef thrombin is used with Gelfoam. The hemostatic action of the combined Gelfoam and beef thrombin is good, as shown by Light[10] and Pilcher and Meacham,[9] and there is as yet no reported antigenicity of human beings to the beef thrombin.[11]

We have now used Gelfoam in a limited number of varied and selected cases, in an attempt to arrive at a preliminary evaluation of it. We used it following two cystectomies, four impacted lower third molars, one apicoectomy, and six extractions. In all of our cases we were actually more interested in the problem of filling "dead space" than in effecting hemostasis, although it is true that none of these patients had secondary bleeding, and from clinical observation it seems to us that there was a very minimal amount

*The Gelfoam used in these investigations was supplied by the Upjohn Company, Kalamazoo, Michigan.

of postoperative oozing. Our technique in using Gelfoam was the same in the cases of cyst, impaction, root amputation, and some extractions. Following the surgical procedure, a piece of Gelfoam, soaked in thrombin and of sufficient size to fill the cavity, was placed in the area. Any flaps present were now sutured into place. Postoperative results, as judged by the patients' symptoms and recuperative course, and our own clinical observations, point to more rapid healing than usual and lessened edema. Wounds do seem to fill in more rapidly than without the "artificial clot," and there is little need for postoperative care.

In the case of one routine extraction, there was very little bleeding from the socket, and we used thrombin-soaked Gelfoam in the hope of providing an artificial clot that would prevent osteitis. Even a slight amount of blood flowing from the base of a socket or from the soft tissue margin can be drawn into the meshes of the gelatin sponge, thereby forming a blood clot to fill the cavity. Further investigation of this use of Gelfoam, and its possible application to the treatment of "dry sockets," will be done. In another case, following the removal of an upper six-year molar, we found that the palatal root tip had extended into the antrum, its removal leaving an opening into the sinus. The occurrence is not unusual, and the formation of a clot in the socket usually obviates complications. In this case we used an artificial clot, as it were, of Gelfoam to insure sealing off the antrum from mouth contamination.



1. *2.* *3.*

Fig. 1.—Gelfoam in three forms: (*1*) dry, (*2*) thoroughly wetted, and (*3*) wrung out after being wet.

Our use of Gelfoam in several sockets following routine extractions poses some interesting questions as to the possibilities of future use. In four cases we used our Gelfoam without thrombin, soaking it only in saline solution. The gelatin sponge is in itself slightly hemostatic. As we mentioned before, control of bleeding, although important, is secondary in our estimation to obliteration of space. Using plain Gelfoam will aid in the control of bleeding, and, at the same time, elimination of thrombin cuts down the cost of using the agent—a factor which is to be considered if Gelfoam is to be used for more than just extensive surgical procedures. In our few cases, the saline-soaked Gelfoam was effective in accelerating healing of sockets, and it did decrease bleeding. The possibility of using Gelfoam as a vehicle for the introduction of penicillin, or other saline soluble drugs, into wounds should be considered.

The mechanics of using Gelfoam (Fig. 1) is very simple. The dry sponge, packaged in a sterile container, is cut to the desired size, the thrombin solution is prepared by adding saline to the powdered material, and the sponge is immersed. It is then lifted out, thoroughly squeezed to eliminate air from the meshes, and reimmersed. As it soaks up solution, the sponge returns to its original size. Excess fluid is blotted off by placing the gelatin sponge on a dry cotton one, and the Gelfoam is now ready to be placed in the wound. In neurosurgery, it has been advised to draw blood into the meshes of the Gelfoam with a suction tip. In our work we find this procedure unnecessary routinely, as there is usually free enough bleeding in a mouth wound to fill the gelatin sponge quickly. Once the Gelfoam is placed, it is firmly adherent, and we have not yet experienced difficulty with its retention.

Summary.—We have had no postoperative hemorrhage in any case where Gelfoam and thrombin was employed, and, indeed, bleeding was of a minimal amount. Healing seemed to be accelerated, and postoperative edema and discomfort minimized. Our results in a limited series have been gratifying, and we anticipate the useful application of an absorbable hemostatic agent to oral surgery. The two primary values of such an agent are, first, the obliteration of "dead space," and, second, control of bleeding.

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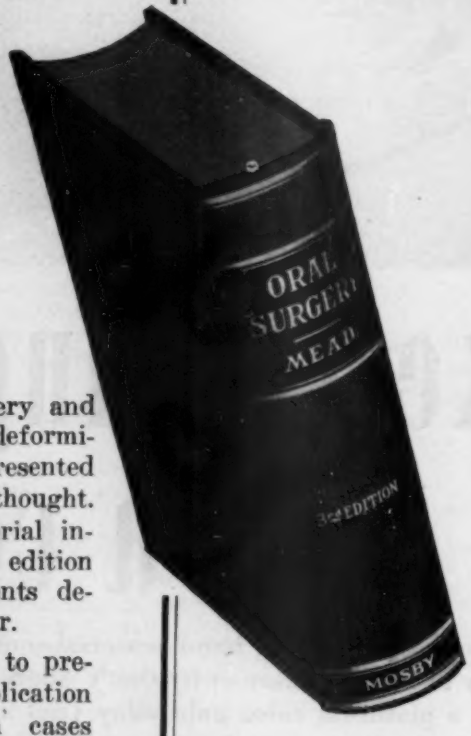
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